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DISEASES OF FRUIT CROPS

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DISEASES OF FRUIT CROPS

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PREFACE

Information concerning fruit diseases of deciduous fruits in the United States is contained mainly in bulletins and circulars issued by the state experiment stations and the U.S. Department of Agriculture and in scattered articles in scientific journals. The only book published on this subject is now thirty-six years old and therefore does not include the many later contributions by workers in this field. It was felt that there was a pressing need for a comprehensive treatment of the subject which would be useful to research workers, teachers, and practicing horticulturists. The comprehensive experience of the author in research and practical field work has prompted this attempt to collect and evaluate the extensive but scattered information on the subject and to present it in a usable form.

An attempt has been made at the same time to present a broader view of the subject than that generally included in bulletins, manuals, and textbooks on plant diseases. Physiological disturbances caused by nonparasitic factors are often as destructive as those due to well-recognized parasitic organisms. In so far as these nonparasitic diseases possess definite symptoms, they are included. No attempt has been made to include insect injury except where the secondary effect of such injury leads to a definite abnormal growth condition.

Citrus and other definitely subtropical fruits are not included. The recent publication of Fawcett on "Diseases of Citrus Fruits" covers this crop.

Fruit diseases occurring in foreign countries, but not in the United States and Canada, are given scant attention unless they are of major concern in the countries where they occur. Admittedly, the viewpoint and experience of the author account for major emphasis to conditions as they exist in the United States and Canada. A number of minor diseases have been omitted, especially if their identity is not well established.

The illustrations have been selected mainly to show symptoms of the diseases. Illustrations of morphological details of the organisms have been used sparingly, since most of them (or those of related species) can be found in any textbook on general plant pathology or mycology.

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In the main, the illustrations are mine; a number were obtained through colleagues, to whom I wish to express my great appreciation.

I wish to acknowledge the helpful assistance given me by many of my colleagues, especially during the preparation of the material on diseases with which I was unfamiliar. I am especially indebted to Dwight Powell for his painstaking reading of the manuscript and his many helpful suggestions; to H. H. Thornberry for his careful review of the virus diseases of stone fruits; to M. B. Linford for his contribution to the section on nematodes on fruit crops.

Among those who have given advice on various disease problems or furnished illustrative material are Ralph Shay, J. C. Dunegan, G. W. Keitt, W. M. Banfield, H. E. Thomas, E. E. Wilson, M. T. Hilborne, J. R. Kienholz, W. B. Hewitt, and S. J. Du Plessis. To K. J. Kadow, formerly Associate Pathologist in the Illinois Agricultural Experiment Station, I am indebted for aid in the initial preparation of the material on some of the diseases.

Harry Warren Anderson

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CHAPTER 1

INTRODUCTION

An attempt is made in the following pages to collect and summarize in a single volume the vast amount of information on fruit diseases which has accumulated mainly during the past fifty years. No attempt is made to acknowledge the contributions of the scores of investigators who have patiently toiled to fill in the many gaps in our knowledge of the cause and control of these diseases. In general, only well-established facts are recorded, but in order to give the student a viewpoint, theoretical discussions are sometimes presented.

The average annual income from the fruit crops of the United States exclusive of citrus fruits is about 1 billion dollars. Experience has proved that most fruit crops unprotected against disease are a total loss except in especially favored localities. It is safe to state that were no control measures employed, diseases would reduce this billion-dollar income to less than 200 million, or a loss of 800 million dollars annually.

Diseases and insects have been very important in the evolution of the fruit industry of this country. In pioneer days the absence or scarcity of destructive insects and diseases permitted every farmer to produce sufficient fruit for his own use, and many city dwellers had sufficient vacant space for a few trees, vines, and bushes in their own gardens. Such commercial orchards as existed produced fruit for the larger, crowded cities. The most serious loss to fruit at this time was not in the orchard but in the fruit bin used for storage.

As the country became more densely populated, commercial fruitgrowing became more profitable. At the same time the disease and insect problems multiplied. Some insects and parasitic fungi were imported from abroad, but many already existed in this country on wild hosts. Crab apples, hawthorns, wild brambles, and grapes are closely related to the domesticated varieties, and the fungi and insects common on these hosts soon found their way to the neighboring orchards and vineyards. With increased transportation facilities the spread of the parasitic organisms was facilitated. A disease once established in an area was certain to be distributed throughout the country. By the middle of the nineteenth century, it was recognized that complete crop failures could be attributed to a single disease such as apple scab or bitter rot.

During the last decade of the past century a decided change in fruit production occurred. Control of diseases and insects by spraying was initiated, and the commercial grower was no longer at the mercy of these pests. On the other hand, the rapid increase in diseases and insect pests rendered the production of fruit on the farm and home orchard still less profitable. This was especially true after the general spread of San José scale. At the same time the commercial orchardist was aided by a better transportation system and the introduction of cold storage for fruit. The consumer now demands fruit of high quality and free of blemishes. This has made it difficult for the farm orchardist to dispose of his surplus crop in years of abundant yield. The net result of all these factors is that during the past forty years the production of fruit has more and more become a specialized industry in the hands of orchardists skilled in the art and science of fruitgrowing. These growers have looked more and more to the experiment stations and Federal Department of Agriculture for guidance in the production of marketable fruit.

It is a curious but logical fact that in spite of a rapidly increasing population and an increased demand for fruit, there has been a decided decrease in the number of deciduous fruit trees in the United States during the last thirty years. With this marked decrease in the number of trees, there has been only a slight decrease in total production, and it is probable that there has been an increase in fruit which actually reaches the ultimate consumer. It is evident that diseases and insects have eliminated millions of fruit trees and that those which remain have been made much more productive owing to the control of pests. This more efficient and more stable production in spite of the increase in the number of parasites has been made possible mainly by the scientific development of plant protection. Efficient plant protection has been made possible only by a thorough knowledge of the nature and cause of the diseases of plants.

The method of presentation is designed to aid the reader in finding with reasonable facility the facts he wishes to obtain. The diseases are treated according to host, and the host plants are arranged in the usual horticultural order, that is, pomes, drupes, brambles, grapes, bush fruits, and strawberries. A brief treatment of cranberry and blueberry diseases is added.

The diseases under a given host generally are arranged according to their importance. In order to avoid repetition, diseases occurring on more than one host are treated under the host on which they are most destructive, and reference is made to this discussion in the treatment of the diseases of the other hosts.

Disease Defined. A plant is said to be diseased when it does not develop in an expected normal manner considering the environment in which it is placed. As defined by Heald in his "Manual of Plant Diseases," " . . . disease in plants is any variation from the normal, as expressed either by the checking or by the interruption of physiological activities or by structural changes which are sufficiently permanent to check development, cause abnormal formations, or lead to the premature death of a part of a plant or of the entire individual." This broad conception of disease in plants will be used in the treatment which follows. From this definition it is evident that definite disease symptoms may be caused by agents other than parasitic organisms. For example, a peach tree sprayed with lead arsenate not properly processed may lose part of its foliage and show dead areas in the leaves and brown lesions on the twigs around the buds. These manifestations of abnormal conditions are pathological and will be treated as diseased conditions in this text.

CAUSAL AGENTS OF PLANT DISEASES

The following brief outline of the causes of diseases is introduced mainly for the benefit of readers who have not had training in the field of plant pathology. Diseases are caused by (1) nonparasitic agents, (2) parasites, and (3) viruses.

Nonparasitic diseases may be due to temperature or water relations, injurious atmospheric impurities, mineral deficiencies or excesses, and toxic chemicals applied in control practices. An example of a disease condition produced by low temperatures is the appearance of *frost rings* on apples following a freeze which was not sufficiently low to kill the blossoms outright (Fig. 1). Sun scald of exposed fruit is common following high temperatures and intense light. Deficiencies in soil moisture during critical growth periods may result in stunted growth. In industrial areas chemical pollution of the atmosphere with noxious gases such as sulfur dioxide may cause extensive damage. Within recent years extensive experimental evidence has indicated that mineral deficiencies or excesses cause well-defined diseases. This is especially true where some of the minor elements such as boron, zinc, and manganese are deficient or in excess. Symptoms of disease are also evident when some of the common fertilizer elements such as potassium, phosphorus, or nitrogen are not present in sufficient quantities in the soil or are rendered unavailable. Toxic chemicals applied as sprays or used for fruit set or coloring may cause serious damage to the fruit or foliage (Fig. 2). For example, Bordeaux mixture applied during the early growth period to apples may result in extensive russeting of the fruit. Lime sulfur retards growth when used throughout the season on apple trees.

Unfavorable storage and preharvest conditions often give rise to nonparasitic diseases of great economic importance. These include such diseases as soft scald, Jonathan spot, and bitter pit of apples and various types of internal breakdown. Some of these appear only in storage, while others develop in the orchard previous to harvest.

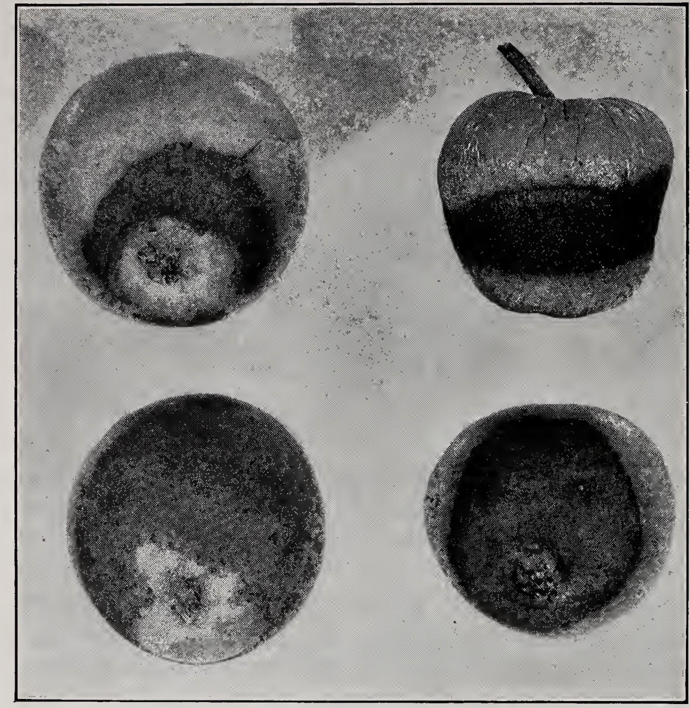

Fig. 1. Frost rings on mature fruit following early-frost injury.

Parasitic Diseases

This group includes those diseases incited by living organisms. Most fruit diseases are due to attacks by *bacteria*, *fungi*, or *nematodes*.

Bacteria. Bacteria are minute single-celled organisms which multiply by simple transverse splitting of the elongated mother cell (fission). All bacterial diseases of fruits are caused by the rod-shaped group of bacteria, and none of these pathogens form spores (endospores). Bacteria are classified also on the basis of whether or not they are motile or nonmotile (Fig. 3). Most of those responsible for fruit diseases are motile.

Fungi. Most fruit diseases are incited by *fungi*. Because of the complexity of the life cycle of these organisms, a detailed description is given of some of the principal groups, together with the terminology employed in the treatment of the specific diseases. This will enable the reader to understand the technical descriptions, in case he wishes to acquire a better understanding of the disease in question.

A fungus "plant" usually consists of a number of branched threads called *hyphae*. The term *mycelium* is used to indicate the mass of threads produced by the growth of the hyphae. The hyphae may be divided into cells by cross walls or may be undivided. The hyphae give rise to spores or spore-bearing bodies. The spores may be compared to the "seeds" of higher plants in that they play an important part in the reproduction and dissemination of the fungi and often are the means of allowing the fungus to withstand unfavorable periods for its development or survival. Classification of the fungi is based largely upon the type of spore or fruiting body produced by the fungus.

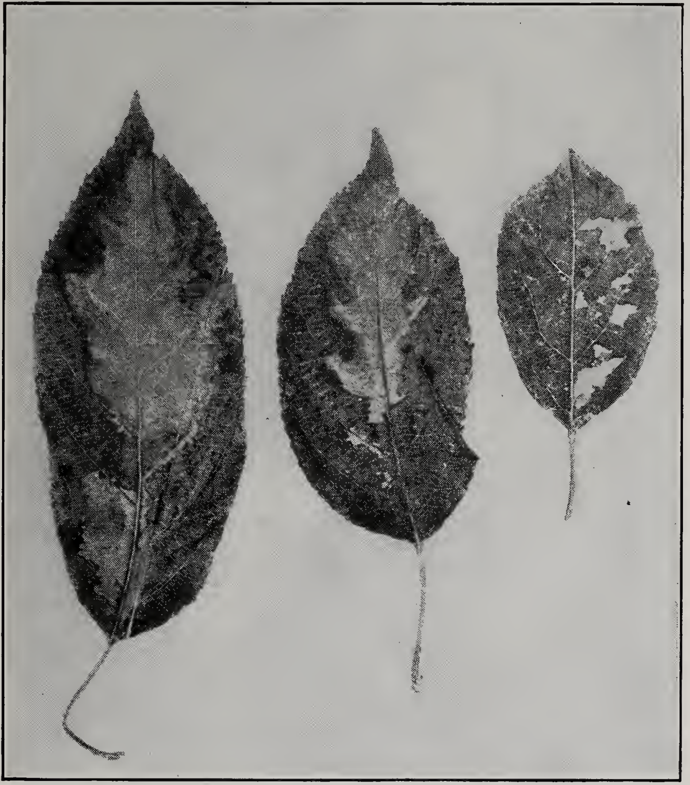

Fig. 2. Sulfur burn on apple leaves following high temperatures.

Since fungi as well as bacteria lack chlorophyll, they must obtain their food mainly from organic substances elaborated by the chlorophyll bearing plants, either directly from the living plant, in which case they are *parasites*, or from decomposing organic material, in which case they are *saprophytes*. Many of the parasitic fungi have periods in their life cycle when they may lead a saprophytic existence; for example, the apple scab fungus passes the winter in dead apple leaves where it continues to grow and produces the spores which bring about infection in the spring. Other fungi called *obligate* parasites are dependent entirely on living tissues, although they form resting spores to tide them over unfavorable conditions.

The fungi with which we are concerned fall into four main classes: (1) Phycomycetes, (2) Ascomycetes, (3) Fungi Imperfecti, and (4) Basidiomycetes.

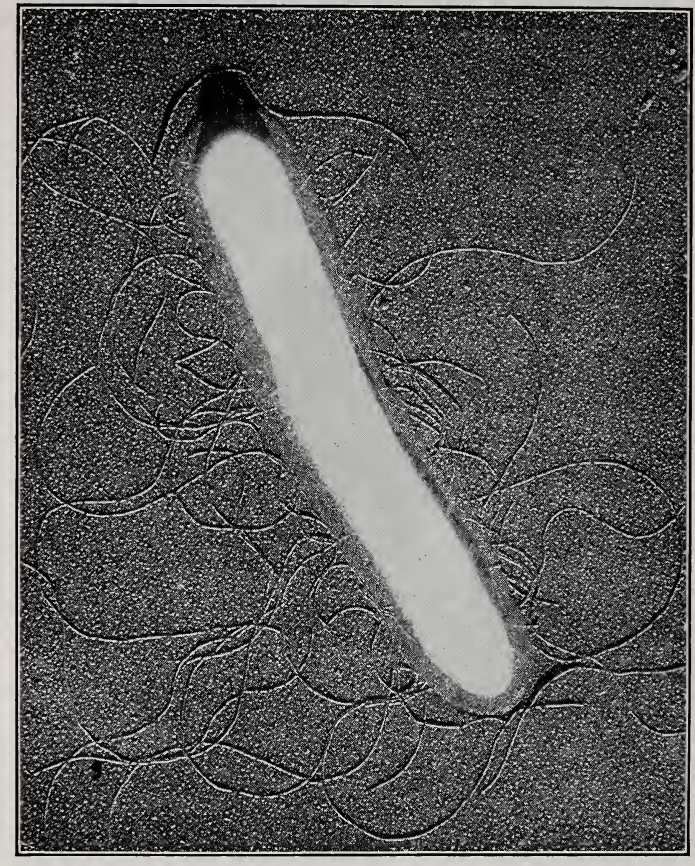

Fig. 3. Typical bacterium with peritrichous flagella. (*Photograph by the University of Illinois Electron Microscope Laboratory.*)

Phycomycetes are characterized by the absence of cross walls in the multinucleate vegetative hyphae. In most cases a sexual spore is formed. The "downy mildews" and "bread mold" are examples of this group.

Ascomycetes have septate (cross-walled) hyphae and produce their sexual spores in a saclike structure, the *ascus*. These spores are called *ascospores* and are very important in that they are usually the winter spore stage enabling the fungus to survive unfavorable conditions. The asexual spores, *conidia,* are usually produced during the parasitic phase on the host plant. Various types of sexual and asexual *fruiting bodies* are produced in this group, and the character of these is the basis of the classification into orders, families, and genera. The sexual fruiting body when present is called an *apothecium* when the ascus layer is exposed, and a *perithecium* when the asci are enclosed by the wall of the fruiting body. The conidial fruiting body, if present, is either an *acervulus* with the conidia exposed, or a *pycnidium* if the conidia are borne inside a protecting wall. In some cases no fruiting body is formed, and the conidia are produced on stalks (*conidiophores*) arising from the mycelial mat. When the conidiophores arise in distinct masses, the individual groups are called *sporodochia,* or *coremia.* Ordinarily the conidia do not survive the winter and are therefore spoken of as the summer spore stage. The ascospores usually are responsible for *primary infection* during the growing season (Fig. 4).

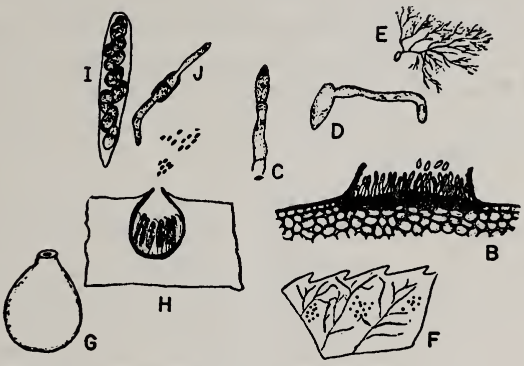

FIG. 4. Life history of the apple scab fungus. *B,* section of lesion of imperfect stage; *C,* conidiophore and conidium (summer spore) of scab; *D,* germinating conidium; *E,* mycelial growth developing from conidium; *F,* perfect stage on leaf, black dots representing the perithecia; *G,* single perithecium; *H,* cross section of leaf with the embedded perithecium showing asci and ascospores, with ascospores being discharged into the air from the perithecium; *I,* single ascus with eight ascospores; *J,* germinating ascospore.

Fungi Imperfecti is a name applied to a group of fungi which, for the most part, are similar to the Ascomycetes, except that they either do not produce an ascospore (perfect) stage, or this stage has not been discovered. A few fungi of unknown affinity are also placed in this class.

Basidiomycetes, to which the rusts, smuts, and common mushrooms or toadstools belong, is characterized by the production of a *basidium* on which ordinarily four *basidiospores* are produced. The basidia in the mushrooms consist of club-shaped cells borne in a solid layer (*hymenium*) on the gills or pores of the fruiting body, the "toadstool." The basidiospores are formed on four short prongs, *sterigmata,* on the basidium.

The *rusts* usually have a rather complicated life cycle. In the full life cycle of rusts such as the black stem rust of wheat, there are five spore forms: (1) pycniospores, (2) aeciospores, (3) urediospores, (4) teliospores, and (5) basidiospores (sporidia). The rusts causing fruit diseases are mostly short-cycle, that is, lacking one or more of the five spore forms. All the spore forms may occur on one host (*autoecious*), or one or more spore stages may occur on a different host (*heteroecious*). Apple rust, for example, has the telial stage on the common red cedar, with the pycniospores and aeciospores produced on the apple (see Fig. 18, page 56). The teliospores on germination produce a four-celled stalk from which the basidiospores arise. This short stalk corresponds to the basidium of the mushrooms, but differs in that it is cylindrical in shape and is divided by cross walls into four cells. A further understanding of the role of the various spore forms may be obtained by consulting the detailed description of the disease cycles of apple rust and the orange rust of brambles. No common disease of fruits is produced by the smuts, so a detailed description of this group is omitted.

Virus Diseases. Fruit diseases caused by virus infections are attracting increasing attention. This is due in part to a better understanding of the nature of viruses and in part to the apparent increase in the number of new virus diseases which have appeared on fruit crops during the past quarter of a century. It is true that some destructive diseases of previously unknown cause have lately been proved of virus origin, but entirely new diseases have appeared with alarming frequency, the origin of which is still unknown.

In a number of respects a virus behaves like a living organism. It is able to infect plants in which it can multiply and cause a definite well-defined disease. It can be transmitted from plant to plant. On the other hand, it cannot be characterized as a living organism as we understand the term. The virus particles which are responsible for the development of the disease are much smaller than bacteria and are able to pass through filters which retain all known organisms. Unlike most bacteria and fungi, they cannot be cultured in the laboratory and they multiply only in the presence of the living cells of the host.

Virus diseases are especially important and prevalent on stone fruits (peach, plum, cherry, apricot, and almond) and on the brambles, especially raspberries. Up to 1930 only five virus diseases of stone fruits were known, since which time over forty new ones have been reported.

Nematodes.[1] Nematodes that attack plants are minute slender worms that rarely exceed 1/20 in. in length. Because of their shape and the way they move, they sometimes are called eelworms, especially in British literature. The special study of nematodes is *nematology*.

Nematodes are of many types with very different habits. The soil contains a great many harmless kinds that feed upon microorganisms and decaying organic matter. A few kinds are beneficial in that they kill and feed upon harmful kinds. The nematodes that cause disease in plants are obligate parasites, requiring living plants from which to obtain their food in order to grow and reproduce. A few kinds, including *Aphelenchoides* that cause disease in strawberry plants, can be reared in the laboratory in cultures of fungi, but the majority can be reared only in or on those parts of plants where they can establish their usual parasitic relationship. These nematodes are related to the roundworm parasites of animals, but no species is known that can develop in both plants and animals.

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[1] This section was prepared by M. B. Linford, Professor of Plant Pathology, University of Illinois. Professor Linford aided also in the preparation of the material on nematode diseases under the various fruit crops.

This discussion will be limited chiefly to a few kinds of nematodes that cause important diseases in fruit crops. These include two genera that are chiefly parasites of roots and one genus that develops between the young leaves in the central bud of the strawberry plant. Nematodes may be *endoparasites*, feeding chiefly inside the plant, or *ectoparasites*, feeding from the plant surface. There are many ectoparasites of roots and also endoparasites of leaves, stems, and even floral parts that must be omitted from this discussion. Even the endoparasites may live part of the time in soil around roots and live over winter or in fallow fields between crops either free in the soil or in remains of old plants.

Morphology and Life Cycle. Despite their small size, these nematodes are complex animals that have well-defined internal organs with special functions. The surface of the somewhat cylindrical body is covered with a tough, nearly transparent cuticle through which many details of internal structure can be observed when the living or suitably prepared nematode is examined under a microscope. There are well-developed muscles that enable the nematode to propel itself, and others with specific functions such as those that enable the nematode to suck in its food. The nervous system, although difficult to observe, enables the nematode to respond to certain stimuli and, importantly, to recognize sources of food. The alimentary tract extends in a straight line almost from end to end of the body, but it includes remarkably effective mechanisms for ingesting food and has associated with it special glands that secrete digestive fluids. Reproductive systems are relatively simple but highly efficient.

The mouth, located centrally at the anterior end, is equipped with a sharp-pointed, hollow *spear,* or *stylet,* that the nematode can protrude forcibly beyond its lips. To obtain food, the nematode drives this stylet into a living cell, and then, with the aid of a muscular bulb in the esophagus, sucks out cell content as food. At least some kinds of nematodes, after inserting the stylet into a cell, inject a secretion into that cell before beginning to suck out their food. Both this secretion and the withdrawal of cell contents may contribute to causing disease in the plant fed upon. Some kinds of nematodes that move about within roots or other parts of plants as they feed also use the spear to break larger openings in cell walls, enabling the nematodes to pass through these cells, which are killed in the process. Such cell destruction also contributes to disease.

Nematodes *reproduce* by laying eggs. These hatch, releasing young larvae of wormlike form which, with rare exceptions, are ready to begin feeding. Eggs may be distributed singly or a few in a place by nematodes that remain worm-shaped and mobile in the adult female stage. Females of some nematodes, however, lose powers of locomotion before reaching maturity. These lay all their eggs in one cluster, an egg mass. Typically, adult nematodes are of two sexes, but in some species there are far fewer males than females, and in other species males have never been observed if they occur at all. Even when males are as abundant as females, in some species they feed little, if at all, after attaining maturity.

The rate of nematode multiplication, although rapid in comparison with higher animals, is not in the same class as that of bacteria and fungi. Most of the plant parasitic species that have been studied require 3 weeks or more for a generation, egg to egg, and some generally produce only one generation per year. The number of progeny in one generation may be many hundreds. In nature, where mortality from various causes may be high, it sometimes requires several years after a plant-parasitic nematode has accidentally been introduced into a field before it multiplies sufficiently to cause conspicuous disease in large numbers of plants.

Environmental adaptations of various nematodes differ widely according to the meager information available. Among two species of *Aphelenchoides* that cause disease in strawberry plants, one thrives at lower temperatures than the other. Among the several root knot nematodes in this country, one species commonly occurs farther north than the others, presumably because of ability to develop at lower temperatures or because of better ability to survive cold winters. Certain nematodes are almost strictly limited to light sandy soils; some others build up injurious populations more readily in sandy or muck soils than in loams or heavier soils; still others seem to thrive in heavy soils. Many kinds of nematodes are killed by drying to the "air-dry" condition. Other kinds may remain dormant but alive for many years in the desiccated state and, when dry, be more difficult to kill by means of heat or toxic chemicals than when they are moist and active.

Movement and Distribution. Within moist soil nematodes move freely by their own power, but they move slowly and apparently over distances best measured in inches. They are strongly attracted to roots, and once near the roots of one plant they probably rarely wander away and move to another plant. Within a field and from one field to another they may be transported by any agency that moves moist infested soil or plant parts or, with certain species, even dry soil and dry plant debris. Tillage implements and running water, during heavy rains or furrow irrigation, contribute to local spread. Movement over great distances occurs freely with the shipment of infested nursery stock. Unfortunately, such shipment has been going on unimpeded for many years. Plants from an infested nursery may all be infested, with the consequence that no time for local spread and little time for multiplication of nematodes may be required before serious damage may be done to the new planting.

Recognition of Nematode Injury. When nematodes attack a plant aboveground, symptoms may be sufficiently specific so that recognition is not difficult. When roots are attacked, however, symptoms that develop on aboveground parts are rarely, if ever, specific. They are essentially similar to symptoms that may result from destruction of roots or from poor functioning of roots from other causes. They may resemble effects of drought, excessive wetness of soil, or poor nutrition. This makes it necessary to examine roots of suspected plants and often to examine soil from around the roots for ectoparasites. Some nematodes cause fairly specific symptoms on the roots themselves, while others do not. Diagnosis of nematode injury, therefore, commonly requires finding and identifying nematodes of species that are known to be capable of causing the type of disease with which they are associated. Merely finding nematodes is not enough, because the soil contains great numbers of kinds of harmless nematodes that quickly invade and develop in plant parts that have been killed by fungi or by other agencies.

Identification and Classification. Accurate identification of nematode species should be a part of any diagnosis of nematode injury, because it is apparent that species within a genus may differ greatly not only in what crops they can attack, but also in the severity of the disease that they cause in a given variety of one crop. Information as to what to expect from different species is still very limited, however, partly because only recently have even the specialists known how to distinguish species within certain genera. Today there are few nematologists who are prepared to make such identifications, but a rapidly increasing interest in the plant-parasitic nematodes may be expected soon to correct this condition.

Names of genera and species that one finds in all but very recent books and articles concerning nematodes are different from many of the names now accepted. Classification of the order Tylenchida, in which most of the plant parasites belong, was revised in 1949 by Thorne. The important root knot nematodes had been regarded as one species and, for some years, had been called *Heterodera radicicola,* and then *H. marioni,* until 1949 when Chitwood demonstrated that actually there are several species. He removed them from *Heterodera* and reestablished for them the old generic name *Meloidogyne.* In 1952, Allen revised the genus *Aphelenchoides,* but, more relevant to the present discussion, the two species of this genus that attack strawberry were not distinguished one from the other until 1942, when Christie described *A. besseyi.* The genus *Pratylenchus,* which includes the root lesion nematodes and meadow nematodes, was revised in 1953 by Sher and Allen. These workers recognized ten species, three of which they described as new, and reported finding other members of the genus that are still not described. These workers found it necessary to establish an involved synonymy because of many faulty identifications in the past. Especially confusing is the fact that many nematodes have been reported as *Pratylenchus pratensis,* or an older synonym, during a period of years when almost any member of the genus was regarded as being that species. Still worse, for a period of years, *Pratylenchus* and several genera together were called *Anguillulina.* One must be cautious, therefore, about accepting as accurate the species identifications reported prior to dates of generic revision listed in this paragraph.

No general outline of nematode classification is attempted here, but a few important characteristics of three genera are presented, chiefly in relationship to life history and parasitic habits.

The genus *Aphelenchoides* Fischer includes the bud and leaf nematodes. Two of the species develop as ectoparasites among young parts of the growing bud of strawberry. Both species also attack the aerial parts of some other kinds of plants. Both are slender, very active forms in all developmental stages. Males are abundant in both, and they evidently feed. Females distribute the eggs among immature leaves in the bud where hatching occurs and where new generations develop. These nematodes apparently do not live long in the soil away from host plants. For identifying details of the genus and for separation of the species the reader is referred to Allen, 1952 (see Selected References at the end of this section).

The genus *Pratylenchus* Filipjev includes a group of species commonly called root lesion nematodes, or meadow nematodes. For many years they were grouped with other genera in *Anguillulina.* Less active and somewhat thicker-bodied than the foliar nematodes, they remain wormlike in form and continue to move about during all developmental stages. Typically they are endoparasites of roots and other subterranean parts of plants, breaking through cell walls and thereby destroying tissue as they move about to feed. Eggs are distributed through the invaded tissues and perhaps also in soil around roots. Males occur freely in some species but rarely or not at all in others. The necrotic lesions caused by these nematodes are readily confused with lesions initiated by fungi, and these lesions may readily be occupied by either fungi or bacteria. The several species of *Pratylenchus* evidently differ in many important respects as to host range, pathogenicity, and environmental adaptations. Until recently, however, the name *P. pratensis* has been applied almost indiscriminately to members of this genus, with the result that valid data concerning biological differences between species are very limited. For the taxonomy of this genus, consult Sher and Allen, 1953.

The genus *Meloidogyne* Goeldi includes several species of nematodes that cause the widely known disease "root knot." Prior to 1949 these had all been regarded as one species, for many years called *Heterodera radicicola,* and then *H. marioni.* They are highly adapted endoparasites, chiefly of roots and other underground parts, causing distinctive knotlike swellings, or galls.

Young larvae are attracted to root tips, other very young tissue, or fresh wounds, where they penetrate by forcing their way between cells. Unless larvae are very numerous they cause little disturbance of the tissue as they penetrate and move within the root. Each larva then locates in a site suitable for permanent feeding and loses its powers of locomotion. Here pathological disturbances are set up. A few plant cells near the head of the nematode, stimulated by secretions injected by the nematode and by the sucking out of food, grow to large size and assume abnormal form and function. These *giant cells* tend to interrupt flow of nutrients through the vascular system of the root and to bring an ample food supply to the parasite. Other cells surrounding the nematode enlarge moderately, forming the gall, or knot.

The majority of these nematodes differentiate as females, the males being relatively few and, apparently, unnecessary for normal reproduction at least in some species. The female enlarges greatly, becoming flask-shaped or almost spherical, then begins laying eggs. These accumulate in a gelatinous mass at the large end of the body, either enclosed within the gall or breaking out to the surface. One female may produce several hundred eggs and may still be laying after the first eggs have hatched and the larvae from them have started a new generation. When the female dies it does not become a cyst as in species of *Heterodera.* Its body wall is still white, and essentially no eggs are retained within it, in sharp contrast with species of *Heterodera* in which the body remains packed with eggs protected by a darkened, resistant body wall. Lacking the resistant cyst stage, root knot nematodes are more readily killed by desiccation and by soil fumigants than are species of *Heterodera,* yet they have the ability to develop in so many different host plants that controlling them by crop rotation is difficult.

The morphological differences that distinguish species of *Meloidogyne* are obscure and difficult to determine. Distinguishing between the species is important, however, in relationship to plant disease because of differences in host range and of severity of attack on common hosts. As a group, these root knot nematodes are able to parasitize over 1,800 different species of plants. Although the host range for any one nematode species is not yet fully known, probably each one can develop in many plants; yet there are some differences in host range that are important in relationship to crop rotations for nematode control. It is also important that crop varieties selected for resistance to one species of *Meloidogyne* may be susceptible to another. Some examples are presented in discussions of root knot of stone fruits and of strawberry.

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HOW TO IDENTIFY A DISEASE

Fruitgrowers, extension horticulturists, and agricultural agents are often confused by the numerous types of injuries which occur on fruit crops. In some cases only a specialist with extensive experience in fruit pathology is able to identify a specific disease, but in the great majority of diseases an inexperienced person by careful observation can identify the malady with sufficient accuracy to enable him to turn to the detailed account of the disease in the following pages. As in medicine, the main reliance is on the *symptoms* expressed by the sick plant, especially where laboratory facilities are not available.

Symptoms of Disease. The first question to settle is whether the abnormal condition observed might be due to mechanical injury, or by insects or other animals. For example, a tree with yellow or off-color foliage may be girdled below the ground line by mice. Curling of the leaves or warty fruit may be due to plant lice injury. The next question is whether the "sick" condition is confined to a few trees, bushes, or vines in the plantation or whether it is uniformly distributed or at least present on a number of plants. If found generally distributed, the causal agent is likely to be bacteria, fungi, or adverse climatic factors. If localized, one should be suspicious of root trouble, especially if the whole plant shows signs of weak growth or off color. In the case of a wide distribution on the aboveground parts of the plant, the following outline of symptoms should give a clue to the identity of the disease.

Color Changes from Normal. If the whole plant is involved without evidence of leaf spotting, the trouble is likely to be of physiological origin, that is, due to root or stem (trunk) injury or improper cultural methods. An examination of the trunk and root system may reveal the presence of dead roots or trunk cankers. (See below for differentiation of root rots and stem cankers.) Some virus diseases such as peach yellows may cause yellowing of the foliage, but this is usually accompanied by other more definite symptoms, as described below.

Wilting and Blighting of Shoots or Entire Plant. Blighting of the terminal shoots of pear, apple, and quince followed by blackening of the leaves is a definite symptom of "fire blight." This may be preceded by "blossom blight," which may not be noticed by the observer unless it is very pronounced. Terminal wilting of new shoots of raspberries is characteristic of *Verticillium* wilt of this crop. In peach, the blighting of single branches is often found to be due to a dead area encircling the limb below the blighted portion, which may have resulted from winter injury or a brown rot canker.

Leaf and Fruit Spotting. Most important fruit diseases show evidence of either leaf or fruit spotting or both. The type of spotting is therefore of great importance in diagnosing the disease concerned. Some of the points to observe are the *color, size, shape* (angular or circular), *border* (definite or indefinite in outline), *effect on the tissue* of the leaf (whether killed or alive), *character of the surface* (presence or absence of a velvety growth or definite minute black spots on upper or lower surface of leaf), and *location* on the leaf or fruit. (In the case of rot spots on the fruit, see symptoms under Rots.) One should also observe whether the diseased spot on the leaf is cut out, resulting in a "shot-hole" effect, and whether there are accompanying symptoms such as yellowing of the diseased leaves or defoliation (see below). A few examples of the use of the above outline follow. In May or early June small yellowish to orange-colored spots are observed on the upper surface of apple leaves. The tissue of the leaf is not killed below the spots. An orange-colored exudate near the center of the spots may be present. There is a thickening of the tissue on the undersurface of the leaf, and this has a distinct yellow color. Observation of the fruit may or may not show similar orange-colored spots near the calyx end. Glancing over the description of symptoms of apple diseases in the text one quickly determines that the disease in question is apple rust. Small, angular, dark purple spots on peach leaves with the tissue killed and subsequent yellowing and dropping of the diseased leaves would indicate "bacterial spot." Here one might confuse this disease with spray injury. Spots due to spray injury are larger, more nearly circular in outline, and do not show the deep-purple color of the bacterial spot. Usually spray injury also shows as a marginal or tip burn as well as a spotting. "Cherry leaf spot" may be easily diagnosed by the definitely circular outline, a tendency for the diseased tissue to drop out, giving a shot-hole effect, the presence during damp weather of small masses of cream-colored spores on the undersurface of the spot, and finally yellowing and dropping of the diseased leaves.

Defoliation. A number of leaf diseases result in prompt dropping of the leaves (defoliation). Sometimes a comparatively light leaf infection will start defoliation. It is evident that some toxic substance is produced by the fungus, which causes a premature formation of the abscission layer at the junction of the petiole with the twig. Yellowing of the leaf blade may or may not occur previous to defoliation. Anthracnose of gooseberry and currant, bacterial spot of peach, *Fabraea* leaf spot of pear, and cherry leaf spot are characteristic examples of defoliating diseases, while apple scab, fire blight of pear and apple, and anthracnose of brambles are examples where defoliation is either absent or delayed until the disease is well advanced.

Defoliation may result from the use of the wrong type of spray material. For example, peach trees sprayed during damp weather with Bordeaux mixture will drop most of the leaves within a few days after the application.

Malformations. Swelling of shoots or leaves, galls, excessive production of shoots, etc., indicate specific diseases, although as with other symptoms these malformations may be due to several agents. For example, galls are often the result of attacks by gall-forming insects. The uniformity in size and shape of the insect-produced galls in general distinguishes them from those caused by parasitic fungi and bacteria. Crown and cane gall on brambles caused by bacteria are easily recognized, but this same disease on apples is often confused with callus formation at the graft union which is not of parasitic origin. Excessive enlargement, thickening, and curling of peach leaves are characteristics of "peach leaf curl" caused by one of the Ascomycetes, while a closely related species causes the "pockets" disease of plums, where the fruit is distorted in shape and is often swollen to ten times the size of a normal fruit. Excessive production of hairy roots on the apple is caused by a bacterium closely related to the crown gall organism. Root swellings, galls, and knots, together with the excessive production of fiberlike roots in the apple, is caused by the attack of an insect, the woolly aphis, and should not be confused with crown gall or hairy root.

In some cases the swellings are composed of a combination of fungus and host tissue. This is true of "black knot" of plum, where elongated black swollen areas occur along the shoot.

Cankers. A canker is a diseased area on the stem or branch, usually well defined and often resulting in the death of the bark within the infected area. The cankered area may be large or small. One should observe such characters as to whether the dead area extends to the wood or is more or less superficial. The surface, whether smooth, rough, or scaly, is diagnostic. Trunk or limb cankers due to fire blight have a smooth surface with a definite crack between the diseased and healthy portion of the bark. Small superficial cankers are produced by the apple blotch fungus on the current season's wood. These are evident for a number of years as rough rings of scaly bark on the older limbs. On some cankers the fruiting bodies of the fungus are to be seen, for example, blisters, or "nailheads," in apple blister canker and pimplelike pycnidia on apple black rot cankers. Dead areas in the bark may be due to winter injury, as, for example, the crotch injury to apple trees following a severe winter.

Rots. Fruit rots are of special importance as fruit diseases because of the enormous losses which result. Identification of a particular fruit rot is based on the following characters: (1) time of its appearance (on immature or nearly mature fruit or only after harvest), (2) association with injury or wounds, (3) average size and location on fruit in case of "spot" type of rot, (4) surface color and presence or absence of fungus fruiting bodies, sunken or even with surrounding healthy tissue, (5) character of flesh in rot spot (watery, soft, firm, or dry and spongy), and (6) shape of diseased area in section (conical, saucerlike, or distinctly superficial).

This group includes a number of "nonparasitic" diseases such as soft scald of apples, water core, and various types of internal breakdown. Often the common name of the disease gives a clue as to its identity, for example, black rot, soft rot, brown rot, bitter pit, etc. These terms are not always meaningful; for example, in bitter rot of apple the diseased flesh is not especially bitter.

Root and Wood Rots. Death and subsequent rotting of the roots result from the attack of certain soil-inhabiting fungi. These diseases are frequently overlooked until the aboveground parts show signs of distress. Light-colored foliage either over the entire tree or on one side indicates the presence of root trouble. In some cases, the injury will be found on the trunk near the ground line rather than on the roots. Winter injury, mice injury, or borer injury should be looked for. The location of the rotted area and whether or not the rot is confined to bark should be determined. If the wood of the rotted root of apple is brittle and punky with a black incrustation, one would suspect "black root rot." If white or black strands of mycelium are observed along the surface or beneath the rotted bark in peach, the disease is likely to be "*Armillaria* root rot." Rotting and blackening of the "core" of strawberry plants definitely indicate the "red stele disease" of this crop.

HOW TO GET DISEASES IDENTIFIED

If trouble is experienced in determining the cause of a particular disease, the grower or county agricultural agent should consult the plant pathologist at his state experiment station. Care in the preparation of the specimens sent will aid greatly in getting prompt service. A generous supply of the diseased material should be collected. This should include, if possible, several stages in the development of the disease. The selected specimens should be wrapped separately in waxed paper, or sealed in a pliofilm bag, and mailed promptly, together with a letter giving the facts as observed by the collector, such as its appearance in the field, whether general or local, difference of severity on varieties, and what spray applications if any had been made.

CONTROL OF FRUIT DISEASES

Only general principles of disease control will be discussed at this point. The reader is referred to the discussion of specific diseases as to methods which are likely to be effective against the disease in question.

Various systems of classifying control measures have been suggested in textbooks or manuals of plant pathology. No attempt is made here to follow any of these systems but rather to discuss the various control measures which are applicable to fruit diseases.

Selection of Resistant Varieties. This method of control applies to both parasitic and nonparasitic diseases. It should be pointed out that resistance to one or several diseases does not imply that this resistance is of any importance to the grower where the diseases in question are not prevalent or are of minor importance in the region where the fruit is grown or where the resistant variety is not adapted to the region. For example, a pear resistant to fire blight but subject to winter injury would be of little value to the northern fruit grower. An apple variety highly resistant to scab but very susceptible to apple blotch would be of doubtful value to a fruitgrower in the region where blotch is prevalent. A resistant variety of poor quality from a commercial standpoint would be of no value to most fruitgrowers. On the other hand, the selection of varieties resistant to one or more of the important diseases of a fruit crop and of otherwise good quality may save the grower the heavy cost of other methods of control such as spraying.

By breeding and/or selection, varieties have been produced primarily for their resistance to disease or adverse environmental conditions, but standard fruit varieties vary widely in their resistance to diseases, and thus it is often possible for the grower to choose those known varieties which are resistant to destructive diseases in his locality. In planning new fruit plantings, one should not only consider the quality of the fruit but also the disease factor.

Control by Exclusion. This method of control is usually employed where a disease is not present in a country, state, or fruit-growing area, but which if introduced may be expected to attack fruit crops. Usually, national or state *quarantines* are set up to prevent the introduction of plants or plant parts which might introduce the diseases. Most states have a *nursery-inspection* service which aids in protecting the grower against the introduction of certain diseases in his locality. This is especially effective in the control of virus diseases which if introduced into a plantation cannot be controlled by other methods.

Eradication. Certain types of diseases can be eliminated from a fruit plantation only by eradicating the diseased plants or parts of these plants before a general spread occurs. Most virus diseases can be controlled only by eradication once they are established in a fruit area. Sometimes eradication may be undertaken on a national scope, for example, the elimination of stone fruit virus diseases such as phony peach. The grower himself can control certain raspberry virus diseases by "roguing," that is, systematically removing diseased plants.

Another type of eradication consists in the destruction of wild hosts in the neighborhood of the fruit plantation. This is especially effective where the fungus attacking the fruit crop has an "alternate stage" in some plant which has no particular value. A good example of this is apple rust which has one stage of the rust on red cedar. The elimination of the cedars in the neighborhood of the apple orchard will completely control this disease.

Eradication or cutting out of diseased parts is effective, especially in the case of canker diseases where the fungus ordinarily produces the spores which spread the disease. Eradicant sprays may be used with certain diseases where the fungus lives over winter in dead parts such as the fallen leaves, in the case of apple scab, or in the dead "brush," as in asparagus rust. Painting fire blight cankers with penetrating bactericidal chemicals is an eradication method used with success in some sections of the country.

Sanitation and Cultural Methods of Control. Sanitation is concerned with the problem of reducing the *spore load* of parasitic fungi in the plantation, in the packing shed, and in the packaged fruit. In some cases this involves only the ordinary operations of pruning and removal and destruction of dead branches or plants from the orchard. Thus in the case of a badly blighted apple tree, all the blighted shoots should be cut out and burned, since these not only may carry the blight bacteria over winter but usually harbor the black rot fungus, which because of its location in the tree will give rise to infection on the leaves in the spring, producing the frog-eye leaf spot. Peach fruit attacked by the brown rot fungus if allowed to remain in the orchard will give rise to the fruiting bodies of this fungus in the following spring and will thus bring about blossom blight and subsequent fruit infection. In the packing shed careless handling of rotting fruit will contaminate the sound fruit in the package, and subsequent spoilage may be expected. A few rotting strawberries in a basket will spread the disease to most of the fruit before it reaches the consumer. To carry out sanitary measures effectively and economically the grower should know the nature of the disease and the hazards involved, otherwise the procedure might prove not only expensive but ineffective.

Cultural practices are linked with sanitation because in some cases they involve the same operation; for example, cutting out diseased branches may be done while thinning out the trees in the regular pruning operation. Thinning out the trees allows free air circulation, and thus quick drying of the foliage, which in turn reduces the chance of spore germination and penetration. Strawberry plants well spaced in the row suffer less from the gray mold rot of the fruit than when the plants are matted. Careless pruning, especially of large branches, sometimes results in infection by wood-rotting fungi. These fungi rarely enter a clean-cut surface even if this is not treated.

Proper attention to drainage may reduce the chances of infection by soil-inhabiting fungi. For example, the red stele root disease of strawberry rarely causes injury in well-drained soil even where the causal fungus is present. In some cases the site of a plantation may be an important factor in disease control.

Protection. Protecting the plant against invasion by disease-producing fungi or bacteria is the most widely used method of control for most fruit diseases. Chemical protection consists in the application of sprays or dusts to the aboveground parts of the plant or in some cases to soil through injection or soaking. With few exceptions soil treatment for fruit-crop diseases is rare, so that the grower depends largely upon the use of fungicidal sprays and dusts for protection. A few spray chemicals kill out (eradicate) the fungus when it has actually invaded the plant. The main function of these chemicals, however, is to prevent the germination of the spores or kill the fungus before it enters the plant tissues.

Many factors enter into the choice of a fungicide for any particular situation. The grower is largely dependent upon the research and experimentation of plant pathologists and horticulturists for determining the proper materials to use and the schedule to follow for any particular fruit disease. The research worker or horticulturist in turn must have a thorough knowledge of the disease cycle of the causative organism, of the conditions under which the growers are operating, and of the probable effectiveness of different fungicides under those conditions.

For many years the standard fungicides were those containing *sulfur* or *copper*. The main task of the research worker and the chemical-spray manufacturers was to find methods of enhancing the efficiency of these two basic fungicidal materials by physical or chemical means without increasing toxicity to the plant. In the case of sulfur it was found that efficiency depended largely on the fineness of the particles. Methods were therefore devised by the manufacturers to produce what is generally known as "microfine" sulfurs. These combined with a wetting agent were very effective in the control of many fruit diseases and were usually nontoxic to the plant. *Lime sulfur*, a chemical combination of lime (calcium hydroxide) and sulfur, was one of the earliest chemically combined forms of sulfur. Copper-containing sprays were always used in chemical combinations, and since free copper is highly toxic to plants, these combinations were of the "insoluble" type. The best representative of the copper sprays is Bordeaux mixture, a combination of lime and copper sulfate in solution. Various other types of "fixed" coppers have been developed for reducing toxicity or for securing greater fungicidal efficiency. Wettable microfine sulfurs, lime sulfur, and Bordeaux mixture are still widely used in fruit-disease control because of their low cost and relative efficiency.

Within recent years entirely new types of fungicides have been developed. These are the *organic fungicides* as contrasted to the *inorganic* copper and sulfur combinations. Since the organic fungicides are usually composed of complex molecules with long names, it has become necessary to designate them by code words such as "ferbam" (ferric dimethyldithiocarbamate). Generally, the organic fungicides are more specific in their actions, that is, are effective against only a limited number of diseases; while the inorganic are more generally useful. However, certain of the organic fungicides are effective over a wide range of diseases and are much less toxic to the plants. Most organic fungicides are more expensive, and in some cases this precludes their use except as emergency sprays.

Organic fungicides as presently used in fruit protection may be classified as (1) *dithiocarbamates* (ferbam, zineb, nabam, ziram, and maneb), (2) *quinones* (dichlone, chloranil), (3) *organic mercuries,* (4) *quarternary amines,* (5), *glyoxalidines* (glyodin), and (6) other organic fungicides, represented at present by a single compound, *captan* (N-(trichloromethylthio)-4-cyclohexane-1, 2 dicarboximide), which promises to be an outstanding fungicide for fruit-disease control. Some of these organic fungicides have been sufficiently tested so that they have a definite place in fruit-disease control; others need to have further tests before they can be evaluated. The grower should inform himself as to their relative merits for his particular locality before attempting to use them on a large scale. *Combinations* of fungicides and insecticides are often recommended in order to save labor. The grower should also inform himself in advance as to what combinations are safe or compatible.

Control by Physical Means. Prevention of spoilage of harvested fruit from the time it is harvested until it reaches the consumer is recognized as an outstanding economic problem in the fruit industry. Retailers regularly figure this loss as from 10 to 25 per cent and add this to the retail price in addition to the cost of handling. Less perishable fruit such as well-graded apples may be figured much lower than highly perishable fruit such as strawberries. This loss has been greatly reduced within recent years by a number of improvements in handling through the stages of picking, packing, transportation, storage, and marketing. In picking and packing every effort should be made to avoid *wounding* and *bruising.* Some rot-producing fungi are unable to penetrate the unbroken skin of the fruit, but readily gain entrance through a scratched, punctured, or cracked surface. *Low temperatures* inhibit or delay spore germination and slow up rotting. While most fruit at present is refrigerated during transit and storage, it is of great importance to reduce the temperature as rapidly as possible at the packing shed and to operate in such a way as to reduce the time between picking and the application of cooling treatments.

Noxious gases are given off by packaged fruits such as apples resulting in various types of "scalds." *Ventilation* and the use of oiled paper for absorption of these gases are standard methods of disease control in transportation and storage.

Grading to eliminate wounded fruit or those with incipient rots aids in reducing loss in transit, storage, and market. A single strawberry infected by the bread mold fungus will spread the disease through the entire basket within 2 or 3 days. *Washing* some fruits in water or water with an added disinfectant will often reduce the spore load on the packaged fruit. High *humidity* sometimes results in a deposit of water on the surface of the stored fruit, thus favoring spore germination. Low humidity on the other hand may result in shriveling of fruit. Fluctuations in temperature in the storage results in "sweating" of the fruit, which favors infection by rot-producing fungi.

Application of any of the above control methods, or combination of them, depends on a careful analysis of the disease situation which confronts the particular fruitgrower or fruit handler. Confronted with the greatly increased cost of labor and transportation, the producer must seek the cheapest method of control, but with the emphasis on quality fruit the "cheap" method may result in a high proportion of culls which must be disposed of at a loss.

Many factors are involved in the effective application of disease-control measures. Successful control can be achieved only when these factors are understood. For example, the application of a superior fungicide may be entirely ineffective if the spraying is done after infection occurs. *Timeliness* not only applies to the spray applications but to other control measures such as eradication and sanitation. Eradicating a canker after the spores are discharged or roguing virus-infected plants after insects have fed on them would be less effective than if these operations were properly timed.

Some New Principles of Disease Control. Present methods of disease control as outlined above may be supplemented in the future by certain new developments in this field. Chemical plant protection was based on the principle that certain chemicals could be applied to the surface of the plant parts in such a way as to be nontoxic to the plant, but would prevent the germination of the fungus spores when they came in contact with the chemical layer or would kill the germ tube or hyphae before entrance was gained. The idea that chemicals could be introduced *into* the plant system to prevent infection or to correct nutritional deficiencies has long been considered by research workers. The practical application of such a method has been the spraying or injection of trees with dilute solutions of metallic salts for the correction of chlorosis or other physiological diseases where certain elements are lacking. Recently the field of *plant chemotherapy* has been developed, especially for the control of certain diseases which do not readily yield to other simpler methods of control. It is believed that these chemicals need not necessarily be fungicidal or bactericidal but might be of such a nature as to impart resistance to the host tissues by certain metabolic changes. Others may be fungistatic, thus serving to delay or reduce the harmful effect of the fungus. Interesting results have been obtained, especially in the field of *antibiotics*, which have been found in some cases to become systemic in the plant when applied externally. The field of chemotherapy is still in the experimental stage.

Another development is the improvement of *soil disinfestants* for the control of soil-inhabiting plant pathogens. Until recently soil disinfestants were used mainly for seed-bed disinfestation or where the value of the crop and the limited area warranted the high cost of the process. Improved methods of application, a better understanding of the problems involved, and greater choice of materials have led to a greatly increased application of soil disinfestants on a large scale for the control of soil-inhabiting fungi, bacteria, nematodes, and insects which attack the roots of fruit crops. Only a few of the many chemicals tested will be treated here. Further reference to their application to fruit diseases will be found under control of root diseases of specific fruit crops incited by nematodes and fungi.

Carbon Disulfide. This is one of the oldest soil fumigants used on fruit crops. After the introduction of *Phylloxera* in the vineyards of France, the French vinegrowers found that carbon disulfide would control this insect when injected into the soil. Thousands of acres of vineyards were fumigated in France between 1880 and 1900. This practice was discontinued after the introduction of *Phylloxera*-resistant rootstock from America. In the United States, carbon disulfide was used mainly as a soil fumigant against insects. It was also regarded as a good nematocide. Its fungicidal property has been demonstrated in its use for the control of *Armillaria* root rot. Unfortunately, its use is dangerous since it is inflammable and explosive. One of its main advantages is that it is fairly cheap and will penetrate the soil to a much greater depth than most other soil fumigants. In orchards a power-driven applicator is used unless a small area is to be treated. A gallon of carbon disulfide will treat approximately 180 sq ft. The soil to be treated should be warm and porous and with a low moisture content but not dry. The applicator should be set so that the liquid is injected at a depth of 6 to 8 in. It is stated that it will penetrate the soil to a depth of 5 to 6 ft. Carbon disulfide is toxic to living roots and should not be injected closer than 6 to 8 ft from living trees or vines.

Dichloropropene-propane Mixtures. This chemical, usually called D-D, is a 2:1 mixture of 1,3-dichloropropene and 1,2-dichloropropane. The fraction toxic to nematodes and fungi is the 1,3-dichloropropene. Its volatility is low enough to make sealing it in the soil unnecessary. It is only a weak fungicide but is quite effective against nematodes. D-D is relatively cheap since in dosages of 200 to 600 lb per acre, the cost would be from $35 to $90, which is sufficiently low to warrant field use for fruit crops.

Ethylene Dibromide (EDB). This chemical has been used very extensively since 1946, especially in the control of nematodes and wireworms. It has a high boiling point, needs no sealing, and thus is adapted to extensive field applications. It is usually marketed in solutions containing a naphtha thinner. It is relatively cheap since, in an 85 per cent mixture, it can be used at a rate of 4 to 6 gal per acre. Ethylene dibromide is considered low in fungicidal properties, but within the last few years it has been found to give remarkable control of certain fungus root diseases. A possible explanation of this is that by the control of nematodes, infection courts for the entrance of fungi are reduced, thus preventing extensive damage to the root system.

The development of machinery for the application of soil disinfestants has made rapid progress in the last few years. There are, however, many factors to be considered in the successful application of any of these materials, but it is safe to predict that soil disinfestants will soon be extensively used by fruitgrowers. [A good review of experiments on the use of soil disinfestation is that of A. G. Newhall, Disinfestation of soil by heat, flooding, and fumigation, Botan. Rev., **21**:189-250 (1955). For details of materials and methods of application, the articles on nematodes in the 1943 Yearbook of the U.S. Department of Agriculture are very helpful.]

CHAPTER 2

DISEASES OF POME FRUITS

The pome fruits—apple, pear, and quince—are grown throughout the Temperate Zones of both hemispheres. Most of the production is in the cooler sections, that is, north of latitude 35 degrees in the United States, Canada, Europe, and Asia. Apples constitute the greater part of pome fruit production, with an average commercial crop of about 100,000,000 bushels in the United States. Pears are a poor second, although in Europe it is one of the leading fruits. Very few quinces are now grown except in a few regions where they are produced for preserves and jellies.

Botanically the pome fruits are members of the rose family (Rosaceae), but botanists differ as to the proper generic names, some preferring *Malus* for the apple and *Pyrus* for the pear, while others use *Pyrus* as a genus for both apple and pear and classify apples under the subgenus *Malus*. The quince is generally classified as *Cydonia oblonga*. The fleshy edible part of the pome *fruit* arises from the floral tube, which becomes fleshy and surrounds the *core* containing the seeds. The core is the developed ovary with the seeds contained in several leathery chambers, the carpels. The fact that the calyx cup does not close at the blossom end of the fruit for some time after the petals fall is important in pest-control measures, especially in connection with codling moth. In some varieties the calyx "tube" remains open throughout the development of the fruit and thus permits entrance of fungi such as *Alternaria*, which later invade the seed chambers and cause a core darkening and subsequent rot.

The pome fruits all originated in southwestern Asia around the Black and Caspian Seas from which they spread over Europe and Asia. The American varieties came to America with the first colonists, who brought seeds of European apples and pears, and from the seedlings selected those which had good quality. Later some varieties were introduced as grafted trees, but with few exceptions the commercial varieties have been selected from chance seedlings which showed up in the home orchards of the early settlers.

Propagation of pome fruits is mainly by grafting, since seedlings of a given variety rarely resemble the parent variety. *Breeding* has been practiced by horticulturists over the past century, and this has given rise to a number of commercial varieties possessing superior qualities.

Extreme variation in the seedlings of pome fruits has given rise to numerous horticultural *varieties.* Whether or not a variety becomes of commercial importance depends on a great many factors, too numerous to go into at this time. One of the important factors is *resistance to disease.* This in turn depends upon the diseases prevalent or important in any given area and the adaptability of the variety to the locality under consideration as well as its market acceptance. Over the years devoted to developing desirable varieties, an unconscious selection for resistance to disease has taken place. The horticulturist, the nurseryman, and the grower try to avoid those varieties which are highly susceptible to destructive diseases, and consequently such varieties are abandoned.

The three species of pome fruits have many diseases in common. Fire blight is destructive to all three, but causes most damage to pear and quince. Scab on apple and pear is caused by different species of the scab fungus but is very similar in its symptoms.

Apple Scab

Early writers called this disease "black spot," or "mildew." The earliest reference to the term "scab" is found in horticultural literature between 1850 and 1860, from which time this term has been generally used.

History. The native home of the apple scab fungus cannot be determined with any degree of certainty from the evidence now at hand. The first botanical description of the scab fungus was by Fries, who collected specimens on apples in Sweden in 1819. Schweinitz, an early American botanist, collected the fungus on cultivated apples in New York and Pennsylvania in 1834. Few references to the disease can be found in horticultural literature before 1860, after which date it was frequently mentioned. In 1869 (apparently a bad scab year) McWhorter of Illinois stated, "Thus it seems a fact that, with the increase in orchards, we have an increase in fungoid infections and insects that infest orchards. It is from this circumstance that several varieties of apples which were most profitable in our first orchards are now so frequently scabby and worthless that they are falling into disrepute." In the same year, Capps, of Mount Pulaski, Illinois, reported, "Much of the fruit is scabby (an unusual thing with us)." If apple scab were native to the wild crab, which is common throughout Illinois, it would seem that scab would have been introduced into the cultivated orchards and would have become generally prevalent long before the dates mentioned.

Early horticultural writers state that seeds, cions, and trees of the various fruits, including apples, were imported into this country from

France and the Netherlands as early as 1650. The scab fungus may survive on twigs and consequently may have been introduced in early colonial times. On the other hand, it may have been native on the wild crab apples and passed from these to the cultivated apple. The fact that the European and American scab fungi are indistinguishable indicates a common origin. It is hardly probable that the scab was native to America and was imported into Europe, but in the light of present evidence it would be unsafe to state that it was imported from Europe.

Geographic Distribution. Apple scab is known to be present in all countries where the apple is grown. In some isolated sections the disease did not appear until many years after the introduction of the apple. For example, in Montana scab was not known until about 1900. Certain sections of that state were entirely free of scab until ten or twelve years later. In irrigated sections with dry summers, scab is rarely seen. Most damage occurs in those regions where humid, cool weather prevails during the spring months.

Economic Importance. Loss from scab over a period of years is greater than that from any other disease of apples. Losses result from (1) reduction in crop, (2) devaluation in grade, (3) foliage loss, and (4) increase in operating expenses. Before the advent of spraying for the control of scab, it was common to find reports of complete failures of the crop over large areas as a direct result of scab. For example, Trelease, in 1884, states that the apple crop over a large area of Wisconsin was a total failure because of scab. In Illinois, the year 1869 was outstanding because of a severe epidemic of scab which ruined the apple crop throughout the state. In 1935 weather conditions were favorable for scab development in Illinois, and all unsprayed trees not only lost their crop early in the spring, but also the first leaves were killed to such an extent that the trees appeared dormant in June.

The first loss in the bearing orchard comes from fruit drop as a result of pedicel infection. In many spraying experiments it has been demonstrated that check plots having no prebloom sprays, but protected against insect attacks, may drop all the fruit by midsummer or earlier. Early fruit drop ascribed by some to failure in pollination and lack of set is frequently really due to scab infection. Some varieties, such as Golden Delicious, while not especially susceptible to scab, may have a heavy pedicel infection and a consequent loss of fruit if weather conditions favorable for scab development occur immediately before the blooming period.

Early loss of foliage occurs when inoculation is extensive enough to give general infection over the leaf surface. Frequently this injury is accentuated by the application of a caustic spray, such as lime sulfur, after the scab fungus has ruptured the cuticle of the leaf in numerous places. This loss of foliage may result in failure of the crop the following season, because the tree is so weakened that fruit buds fail to form. Loss of foliage on young nonbearing trees results in stunting or reduced growth.

The loss to the fruit itself through reduction in grade is considered by the average grower as being the most serious aspect of this disease. Scabby fruit is no longer marketable except as culls. It is not unusual for a grower to cull out 10 per cent of his fruit because of scab alone, even where the usual scab sprays have been applied.

Another important item of loss is the increased cost of production to the grower as a result of the necessity of applying from three to five extra sprays for the control of scab. Spraying is an expensive item in the production of apples.

Symptoms. Scab is usually evident first on the underside of the leaf, but this may depend upon the time of infection. In the opening of the bud the lower surface of the leaf is more exposed and subject to wetting, while at a later stage the upper surface is more likely to be inoculated. If inoculation is delayed, the first scab spots may appear on the upper surface. The diseased area first shows as a faint olive-green spot somewhat darker than the surrounding normal leaf green. When first observable, it is usually ⅛ to ¼ in. in diameter with an indefinite outline. The color deepens with age and becomes almost black, especially on the upper surface. The lesions on the lower surface are often obscured because of the hairy character of this surface and because of a tendency for the growth to be more diffuse. This is especially true when heavy infection occurs and the entire surface is covered with the fungus growth. On the upper surface the lesions are more definite. On young foliage the spots are inclined to have a radiating appearance with a feathery edge, while on older leaves the lesions are more definite in outline. As the infected leaf ages, the tissues in the neighborhood of the scab spot thicken, resulting in an upward bulging of the scab area, with a corresponding cuplike depression on the lower surface. The leaf blade may become curled, dwarfed, and distorted as a result of heavy infection (Fig. 5). The *leaf stem* (petiole) is often infected. The lesions here are extended along the long axis of the petiole but are otherwise similar to those on the leaf blade. When several lesions occur on the petiole the leaf is likely to turn yellow and drop.

Scab spots may remain on the leaf throughout the season without killing all the tissue beneath, but more often the underlying cells die, resulting in a dark brown dead area usually with the black fungus evident in the center. In sprayed orchards the spray material is likely to kill the tissue beneath and immediately surrounding the spot. Such areas are usually lighter in color than those resulting from direct killing of the tissue by the scab fungus.

Scab spots are always velvety in appearance, except where the central fungus growth has been worn off by exposure and renewed growth of the underlying tissues has taken place.

On the *fruit*, early infection frequently occurs on the tips of the sepals and the indistinct grayish lesions are often overlooked because of the hairy covering at this stage. Later, the lesions on the enlarging fruit have the same general appearance as on the leaves, but the spots are usually more definitely defined. Considerable variation occurs, depending upon the time of infection and the varieties of the fruit. In the early stages the spots on the fruit are usually smaller and darker in color than those on the leaves. They soon become almost black, but may vary from a light olive green to a deep brown, depending in part on the abundance of

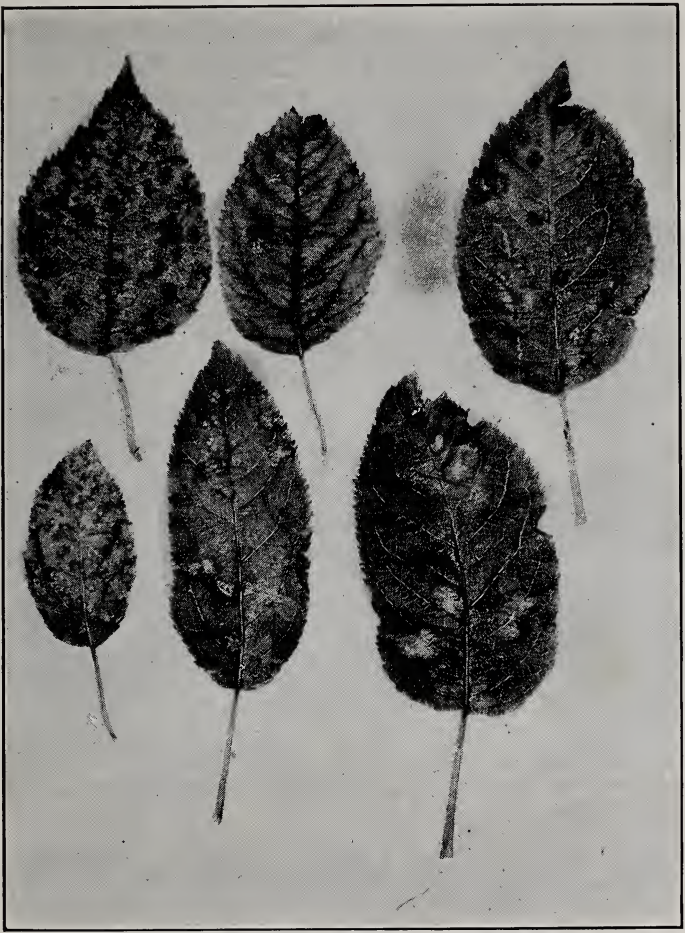

FIG. 5. Apple scab on leaf blades and petioles showing different types of lesions.

spores. The lesions are frequently partly covered by the cuticle of the fruit, which gives a gray appearance to the young lesions, and in older spots the ring of lifted cuticle may be evident at the edge of the infected area. On some varieties, such as the Ben Davis, the scab fungus seems to stimulate the formation of a corky layer beneath the spot, and a warty swelling is produced which seriously disfigures the fruit. In cases of heavy infection early in the season, the fruit is likely to become corky over the entire surface and deep cracks result from uneven growth. This is especially marked where heavy infection occurs on one side, as is commonly the case (Fig. 6). The lesions on the *fruit stem* (pedicel) are much the same in appearance as those on the leaf stem but are inclined to be somewhat broader, especially in thick-stemmed varieties.

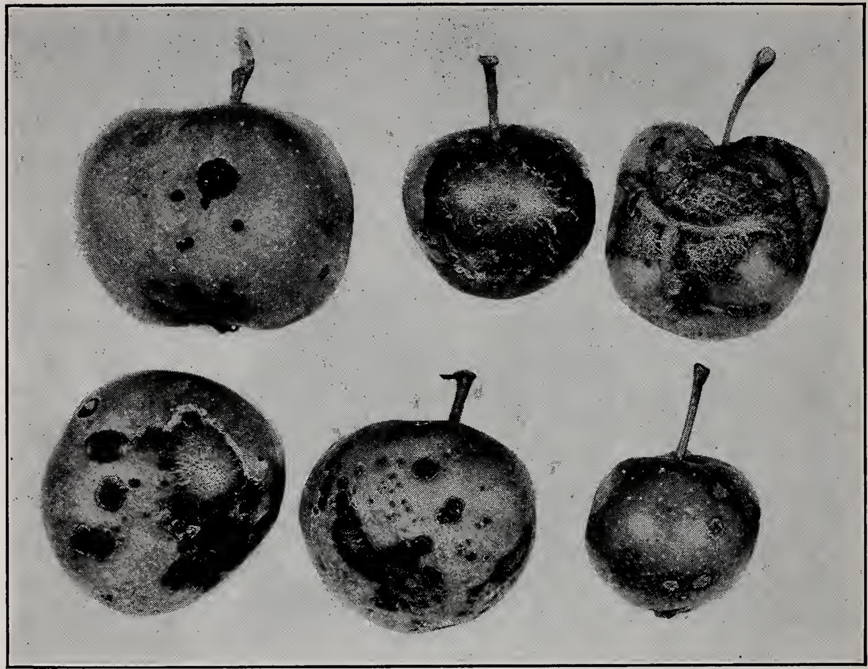

Fig. 6. Apple scab on fruit showing, at lower right, young lesions and, upper right, cracking and distortion.

Scab occurs also on the *twigs* but is not so common as on the organs mentioned above. Under favorable conditions the infections may be so numerous as to cover the entire surface of the terminal growth for a distance of several inches. The individual lesions remain small and usually soon disappear with the growth of the twig. On some especially susceptible varieties a few of the lesions persist throughout the season, sometimes giving the twig a blistery appearance. These lesions are difficult to see and may easily be confused with enlarged lenticels or other malformations. The fungus is found only on the current year's growth.

Causal Organism. The fungus causing apple scab is now known as *Venturia inaequalis* (Cke.) Wint.[1] The imperfect stage has been variously called *Spilocaea pomi*, *Cladosporium dendriticum*, *Napicladium soraureri*, and *Fusicladium dendriticum*. The perfect stage was originally described under the name *Sphaerella inaequalis* by Cooke but was later transferred to the genus *Venturia*.

Hosts. The apple scab fungus is confined to the cultivated apple and certain species of crab apple. It has been determined by cross inoculations by Aderhold in Germany and Palmiter in Wisconsin that the apple scab fungus will not infect species of *Crataegus* (hawthorn), *Sorbus* (mountain ash), or *Amelanchier* (juneberry). The *Fusicladia* on these hosts, as well as the pear scab fungus, are regarded as distinct species. Since Palmiter demonstrated variations in the virulence of selected strains of *Venturia inaequalis* and since there are marked differences in individuals in a collection of crab apple of one species, it is evident that negative results in cross-inoculation experiments are not conclusive. However, the fact seems well established that *Venturia inaequalis* is confined to the species in the apple group of the genus *Pyrus*.

Morphology. MYCELIUM. The scab fungus has a typical septate mycelium which is at first light in color but later turns gray in culture and brownish in the tissues of the host. In young leaves, the mycelium develops in branched, radiating ribbons of parallel strands, thus giving the characteristic dendroid appearance to a young lesion. In older tissues and on the fruit, the mycelial strands are compact and thicker, so that the edge of the lesion is more definite and uniform in outline. The mycelium in the living tissue is located between the epidermal cells and the cuticle. With age it becomes compact and several layers in thickness.

In the dead leaf, the mycelium grows throughout the mesophyll in which it forms a network of hyphae composed of dark brown irregular thick-walled cells. On this network the perithecia are developed.

CONIDIA AND CONIDIOPHORES. The conidia are continuous, ovate to lanceolate, with a truncate base and somewhat pointed apex (Fig. 7). They measure from 12 to 22 μ in length and from 6 to 9 μ in width and are produced in indefinite numbers from each conidiophore. The conidiophores on which they are borne are brown in color, continuous or rarely septate, and vary in length according to conditions. The conidiophores arise from the ribbonlike strands previously described or from the more compact stroma developed later. This stromatic cushion may be ten or more cells in thickness at the center of the scab spot, but near the edge where it forms a wedge between the cuticle and epidermal cells it is usually a single cell in thickness. The stroma is thicker on the fruit than on the leaves.

PERITHECIA (FIG. 7). Mature perithecia are from 90 to 150 μ in diameter, spherical, dark brown to black, with a short beak and distinct ostiole around which are usually several single-celled bristles. The wall is composed of brownish cells, two to five layers in thickness.

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[1] Class Ascomycetes, Order Sphaeriales, Family Pleosporiaceae.

ASCI. The asci in a perithecium vary in number, but as many as 242 have been counted. The usual number is between 50 and 100. The asci arise from a cushion of ascogenous cells at the base of the perithecium, and since they do not all mature simultaneously, both young and mature asci may be found in the same perithecium. The ascus, slightly spatulate in form, is 55 to 75 by 6 to 12 μ with short stipe and thin wall. Sterile organs resembling paraphyses are present when the perithecia are young, but these disappear by the time the ascospores are mature.

ASCOSPORES. The ascospores, eight in number, are arranged in single rank in the upper part of the ascus, but two-ranked in the broader lower portion. When the mature ascus elongates in the presence of water, the spores assume a single-ranked position. The ascospores are unevenly two-celled, the slightly pointed, smaller segment being the one toward the apex of the ascus. They are oval, olive brown in color, and measure from 11 to 15 by 5 to 7 μ.

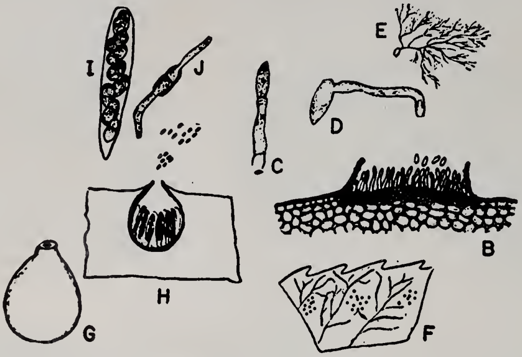

FIG. 7. Life history of the apple scab fungus. *B*, section of lesion of imperfect stage; *C*, conidiophore and conidium (summer spore) of scab; *D*, germinating conidium; *E*, mycelial growth developing from conidium; *F*, perfect stage on leaf, black dots representing the perithecia; *G*, single perithecium; *H*, cross section of leaf with the embedded perithecium showing asci and ascospores, with ascospores are being discharged into the air from the perithecium; *I*, single ascus with eight ascospores; *J*, germinating ascospore.

Disease Cycle. The apple scab fungus has two distinct phases in its life history, one the *parasitic*, or *Fusicladium*, stage, which is the one ordinarily seen on the fruit and leaves, and the other the *saprophytic*, or *Venturia*, phase which develops on the leaves from the time they fall until the following late spring. The first phase is also referred to as the imperfect, asexual, or summer spore stage, while the other is called the perfect, sexual, or winter spore stage. Since the primary infection in the spring is initiated by the ascospores from organs developed during the winter months, it is logical to start description of the disease cycle with the fall of the leaves in the autumn. The saprophytic development begins with the penetration of the fungus into the mesophyll, where it soon forms a network of dark brown hyphae on which the perithecia are later developed

(Fig. 8). The initial growth of the perithecia is made in the fall and early winter. Growth probably continues during warm spells in the winter months, since the perithecia are found to be fairly far advanced by early February of the following year and the young asci are plainly evident. The conditions which bring about perithecial formation are not well understood. It has been established that the apple scab fungus is heterothallic. This fact may account in part for the absence of perithecia under conditions where otherwise they would be expected to develop.

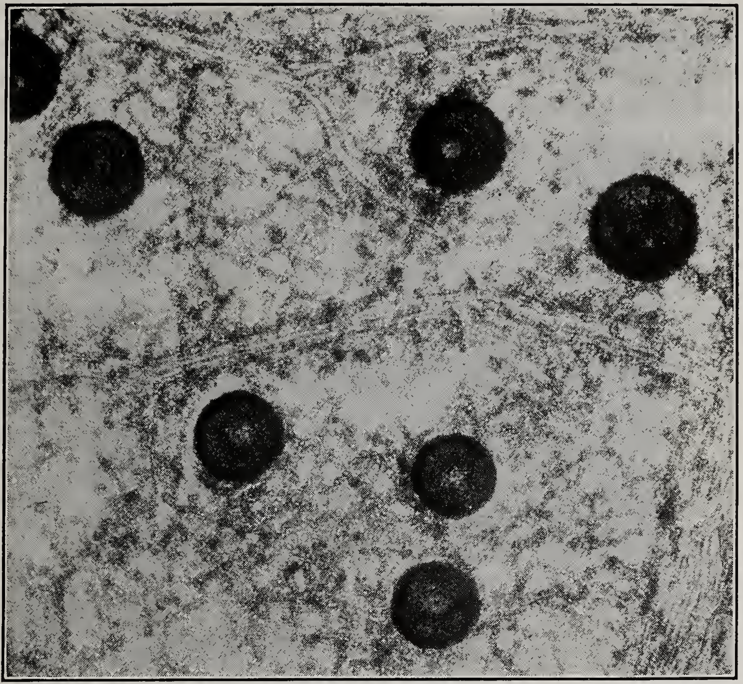

Fig. 8. Perithecia of *Venturia inaequalis* embedded in dead leaf tissue, showing ostioles. Dark hyphal bands from which the perithecia arose are evident in the leaf blade.

Ascospores are usually evident in the asci before the apple buds start to open in the spring. Some of these may show the brown color of the mature stage as early as February, but as a rule the majority of the ascospores in the perithecia mature during the period of the opening of the fruit buds (Fig. 9).

The ascospores in a perithecium do not all mature at once. Young asci may be found in a perithecium even after the first spores are discharged. Perithecia may also be found with no mature ascospores, while others show most of the asci with brown ascospores. In general, however, there is a remarkable uniformity in the time of production of mature ascospores in the perithecia.

Ascospore discharge takes place when the leaves become thoroughly soaked. The asci elongate very rapidly under these conditions, and when a large number of mature asci are present, this elongation pushes off the upper part of the perithecium. If only a few ascospores are mature, the elongated asci extend through the ostiole so that the discharged spores are shot into the air. Asci with immature spores may elongate with or without subsequent discharge of the contents.

Weather conditions play a very important part in the relative abundance of ascospore discharge. The dead leaves must be thoroughly soaked before abundant discharge occurs. During a dry, early spring large numbers of ascospores are matured and remain in the perithecia. Under these conditions a soaking rain will bring about a very heavy discharge, and if weather conditions remain favorable, a resultant severe infection. On the other hand, frequent rains may result in a number of minor spore discharges with an almost continuous period of primary infections.

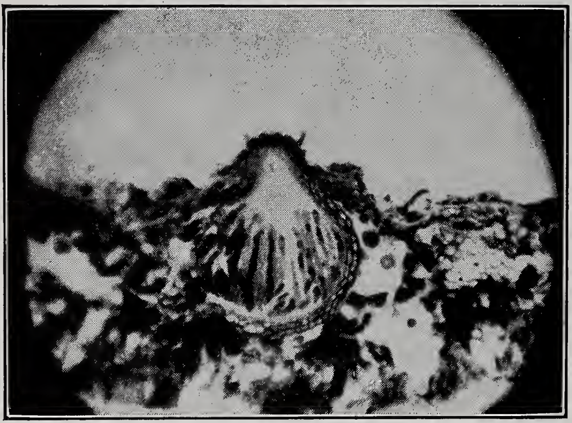

Fig. 9. Section through perithecium of *Venturia inaequalis*, showing asci and ascospores. Note short seta at ostiole.

While some new asci are formed after the first have discharged their ascospores, this process does not continue indefinitely. In general the perithecia are exhausted of viable ascospores within 2 or 3 weeks after petal fall. The total period of ascospore discharge is from 4 to 10 weeks, depending on weather conditions.

The distance to which ascospores are carried by force of the discharge is probably only a few millimeters. This distance, however, is sufficient to carry them into the air currents, which, owing to the minute size of the ascospores, may waft them about like particles of dust. The ascospores after alighting on a surface are not easily removed by washing, since they seem quickly to develop an adhesive material which fastens them firmly to the substratum.

Germination of the ascospores occurs only in the presence of sufficient moisture to keep the spores wet over a period of time which varies according to temperature. At 43°F, for example, the spores must be continuously wet for 13 to 18 hours, at 60° for 7 to 8 hours, and at 70 to 75° for 4 to 6 hours. Under natural conditions, somewhat longer periods may be necessary. The ascospore on germination forms a disklike structure which becomes closely appressed to the leaf surface. From this a very slender tube, or "peg," extends through the cuticle. Between the cuticle and the cellulose wall of the epidermal cells this structure forms a plate of cells from which the mycelial ribbons develop in a radiating manner. The growth of the fungus is entirely between the cuticle and the epidermal cells, but it is evident that a toxic material, or enzyme, is formed which gradually acts upon the cells beneath the superficial growth of mycelium. These cells show a gradual depletion of their contents, often followed by their death.

The period from the time of inoculation to the first evidence of the disease is called the "infection period." In the case of apple scab this may vary from 8 days to over 2 weeks, depending upon the temperature. The conidia are produced even before the scab spot becomes evident to the naked eye. These conidia are formed in enormous numbers and are responsible for the velvety appearance of the scab lesions and their olive-green color. Their outward growth results in rupturing the cuticle.

As long as the conidia remain dry, they are not easily detached from the conidiophores. Contrary to the opinion of earlier workers, the dry conidia are not blown about by wind. Upon wetting, however, they are readily detached from the stalks and then may be washed or blown in mist particles to neighboring leaves or fruit. Like the ascospores, the conidia become firmly attached to the substratum after being in contact with it for a few minutes.

The process of germination and infection is much the same in the two types of spores. Either an appressorial organ is formed beneath the spore in direct contact with the substratum, or a germ tube of varying length is produced, which in turn forms the disklike appressorium from which the infection "peg" arises.

Secondary infection by conidia may occur at intervals throughout the growing season. As a rule, however, infection is likely to be suspended upon the advent of warm dry weather and frequently does not extend much beyond the period of primary infection. This is due in part to the increased resistance of both fruit and foliage and in part to unfavorable weather for scab development.

A second period of scab infection of both fruit and foliage may occur during the comparatively cool weather of late summer and early autumn. Some varieties, such as Grimes Golden, seem to lose their normal resistance as they approach maturity, while in other varieties the increased coating of wax makes them highly resistant. The advent of cool weather in the autumn results in renewed activity of the mycelium of the scab fungus. At the time of leaf fall, penetration into the interior of the leaf and the ramification of the hyphae occur, thus initiating the saprophytic stage.

Two other methods of overwintering of the scab fungus are of possible significance. It is known that on some varieties and in some sections of the country the fungus may attack the twigs and form definite lesions in which the winter is passed. In the spring conidia may be produced, and these may cause primary infection in addition to the ascosporic infection. Most pathologists agree that only rarely in America are infected twigs found, and more rarely are they responsible for primary infections. In England and on the European Continent, twig infection and overwintering of the fungus on the twig in definite lesions are quite common on some varieties and may be responsible for the primary infection.

Conidia may survive long periods when placed under favorable conditions. If they survive the winter after late-fall infection, they might inoculate the opening leaves in the spring. It is probable, however, that such conditions rarely occur in the orchard, since the viable spores would germinate in the fall or winter before the opening of the buds.

The question of the development of scab in storage was thoroughly investigated by Bratley, who concluded that scab lesions may increase in size at any temperature above freezing, but that the increase in size is not of any importance except in those cases where the lesions were not visible at the time of storage and enlarged sufficiently in storage to become visible. Most, if not all, scab developing in storage results from initial infection in the orchard. Fruit kept at ordinary cold-storage temperatures of 32 to 36°F rarely show any evidence of scab increase. In common storage the increase may be marked. All attempts to inoculate mature apples were unsuccessful when they were stored immediately after inoculation. Likewise, all attempts to obtain spread from diseased to clean fruit were unsuccessful. These results indicate that the infections appearing during storage are the result of inoculation while the fruit is on the tree.

Relation to Environment. Apple scab epidemics occur under conditions where cool, damp weather prevails during the early spring season. Moisture is undoubtedly the most important single factor in the process of inoculation and infection. Temperature is important mainly in its relation to the rapidity of drying of plant parts and to the maturing of the host.

Moisture, sufficient to thoroughly wet the old leaves, is necessary for the discharge of ascospores. Free water is necessary for the germination of these spores as well as the conidia after they alight on the leaf or fruit surface. Light rains following relatively dry periods during the early spring are not sufficient to soak the fallen leaves, and consequently few ascospores are discharged unless the rains are either very heavy or continuous over a relatively long period. After the spores alight on the susceptible organs, the period of wetting necessary to bring about infection is dependent upon the temperature. Under optimum temperature conditions (60 to 70°F) infection may take place within 4 to 6 hours after inoculation. Under such conditions it is evident that heavy dews may give sufficient time for penetration of the germ tube. There is no evidence that there is sufficient water on the surface of the leaf to bring about infection in the absence of dews or rains.

Germination of both conidia and ascospores occurs at comparatively low temperatures. Germination of ascospores has been recorded at a temperature near freezing, but the growth of the germ tube is very slow under such conditions. Optimum temperatures for germination of the ascospores and conidia and for infection lie between 50 and 70°F. Infection may occur at 42°. Both germination of spores and infection are decidedly retarded or inhibited when the temperature is maintained at or above 85°F. Temperature also influences the period of inoculation. At 45°F the fungus does not appear macroscopically until 17 days after inoculation, while at optimum temperatures of 60 to 70°F the spots appear within 8 days. High temperatures again influence the rate of development in that the fungus may remain in a dormant condition in the host tissues several weeks during hot dry periods.

High relative humidity does not influence the incidence or development of scab except where actual precipitation takes place as a result of changes in temperature. Heavy snows during the late winter influence the development of perithecia and the discharge of ascospores by keeping the dead leaves moist during the early spring.

Excessively high summer temperatures and extreme droughts do not entirely inhibit production of perithecia and consequent infection the following season. In 1934, regions in Illinois, Arkansas, and Missouri suffered the most extensive drought and the highest temperature in their history. From late March to the middle of August no rains occurred which would permit infection. Temperatures as high as 112°F on 4 days and over 110°F on 28 days in June, July, and August were recorded in western Illinois. Under such conditions, if the scab fungus could not survive extremely high temperatures and excessive droughts, one would not expect it to appear in 1935 in these areas. With 21 days of rain in May, 1935, scab appeared in abundance over the drought- and heat-stricken area of the previous summer.

Light, in itself, appears to have no influence on spore germination or infection. The fact that scab sometimes appears more abundantly in shaded areas of the tree is associated with the longer retention of moisture. Wallace states that the ostiole of the perithecium appears on the face of the leaf which is uppermost and that the perithecia are probably negatively geotropic.

Scab Epidemics. In studying the history of apple scab in the United States, one is impressed with the fact that certain years are noteworthy because of the general prevalence and severity of scab in large geographic areas. These large scale epidemics can be accounted for by a study of environmental conditions. However, no one factor, but a number, must be considered in explaining these outbreaks. A spring of cool wet weather during the opening of the buds may not induce severe scab conditions. This failure under apparently favorable scab conditions may be explained by the general absence of perithecia on the overwintering leaves. Again, an abundance of overwintering perithecia is no index of the possible prevalence of scab if conditions for inoculation and infection are not favorable in the spring months. The factors which are most important in bringing about an epidemic of the disease are (1) conditions favorable for scab development on the leaves of the previous season, (2) conditions favorable for development of the perithecia in the late fall and winter (excessively dry winters with little snow may result in few perithecia in the old leaves), (3) wet weather in early spring (necessary for the production of ascospores and later discharge), (4) continuous periods of wet weather at times when the organs of the tree are susceptible to primary infection, and (5) continued conditions favorable for secondary infection. Widespread epidemics are likely to result when a combination of several of these factors occurs.

Control. Since primary infection is brought about in most cases by ascosporic inoculation, control measures should be directed toward (1) elimination or reduction of perithecial production and (2) protection against infections by the ascospores.

Elimination or reduction of perithecial production may be achieved by growing highly resistant varieties, by destruction of dead leaves, or by applying chemicals which would destroy the fungus on the leaves in the autumn.

Varietal Susceptibility. Since unquestionably there is a difference in the relative susceptibility of varieties of apples to scab, the possibility of the use of existing highly resistant varieties or the future development of such varieties has received serious consideration by students of the subject. Many lists of "resistant" and "susceptible" varieties have been prepared by horticulturists and plant pathologists. Such lists serve their purpose in directing the attention of the grower to the importance of selecting varieties with the object of reducing cost later in spraying, but in the end other factors rightly influence the selection, since a highly resistant variety might be worthless from a commercial standpoint, entirely unadapted to certain environmental conditions or susceptible to other apple diseases.

Selection of resistant varieties is also complicated by certain factors having to do with the scab organism. It has been demonstrated that, like many other fungi, *Venturia inaequalis* has certain biological forms or strains. An apple variety susceptible to one strain may be resistant to another. The presence or introduction of the virulent strain in a geographic section where it had not previously occurred might result in an apparent loss of resistance in the selected variety. Another factor is the well-known fact that some varieties show fairly high resistance under average conditions to fruit infection while the leaves are very susceptible, or the opposite condition may prevail. Furthermore, resistance is often recorded as a result of a limited experience in a limited area. The fact that the plant parts are susceptible at only certain stages of development and that weather conditions are often unfavorable for infection during these periods of susceptibility may lead to a false impression of the resistance of a variety throughout its entire range. Furthermore, certain varieties are known to produce more perithecia than other equally susceptible varieties and so increase the chance of primary infection. Thus, while the selection of resistant varieties is of importance in at least reducing the cost of control by spraying, it cannot be depended upon to eliminate chances of infection. Lists of resistant and susceptible varieties have been prepared by a number of state experiment stations or other agricultural stations. Such lists are of value locally and should be consulted and used by the applegrower.

Control by Orchard Sanitation and Management. Complete destruction of overwintering leaves would eliminate primary, and consequently in isolated orchards secondary, infection. For a number of obvious reasons complete destruction of leaves is not practiced. While clean cultivation, burning, etc., may reduce chances of heavy primary infection, such methods of sanitation cannot be substituted for spraying.

Orchard management is indirectly important in scab control. Attention to planting distances, pruning, and selection of cover crops may aid in increasing the efficiency of the spray program. Proper roads, convenient water supplies, sufficient spraying equipment kept in repair are significant details in orchard management.

Spraying. In the last analysis, spraying must be depended upon for the production of apples free from scab where this disease is prevalent. Since climatic and other conditions vary so greatly in different apple-growing sections, only general principles will be dealt with here. In addition to the effectiveness of the chemicals used, timeliness and thoroughness of spray applications are of paramount importance.

Effective control is obtained by keeping the rapidly expanding organs covered with protective sprays during the period of maximum expansion of highly susceptible tissues and of weather conditions generally favorable for infection. The number of applications necessary to bring about the desired protection can be reduced by careful attention to weather conditions and to a study of the abundance and development of ascospores in the perithecia. Long-range predictions of time of ascospore discharge cannot be depended upon because of local variations in weather conditions. Ordinarily, spray applications are spaced about a week apart during this period, since the rapid growth exposes new tissues which are unprotected if left too long.

The chemicals to be used in scab control depend upon their relative effectiveness as fungicides and their safeness to the plant under the prevailing conditions. In general, under the cool-weather conditions of early spring, sulfur in a finely divided form is regarded as the most efficient fungicide from the standpoint of these criteria. The efficiency of any type of sulfur as a fungicide seems to depend upon the fineness of the particles and its adhesiveness. Lime sulfur is regarded as most likely to cause serious injury to fruit and foliage, but at very high temperatures (90°F and above) any form of sulfur may burn. Copper-containing sprays such as Bordeaux mixture are not only less effective in scab control but are generally regarded as unsafe during the critical period of scab infection. Various organic fungicides have been extensively tested during the past decade. Some of these have outstanding advantages at certain periods. For example, some of the organic mercury formulations give excellent scab control and have the advantage of "burning out" young scab spots. Ferbam, with or without sulfur, is sometimes used since it is also effective for the control of apple rust and apple blotch. Captan is another chemical compound which has become standard.

Dinitro sprays applied to the "floor" of the orchard in the spring before the discharge of the ascospores will destroy as high as 95 per cent of ascosporic inoculum. The use of eradicant sprays reduces the chance of heavy early infection in seasons unfavorable for foliage-spray applications.

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Apple Blotch

The disease now generally known as apple blotch has also been referred to as "fruit blotch," "dry rot," "black scab," "late scab," "cancer," "tar blotch," *Phyllosticta*, and "Phyllostictose."

History. Like many another important plant disease, apple blotch was undoubtedly present in various parts of the United States many years before it was recognized on the leaves of the American Crab apple (*Pyrus coronaria* L.). In 1893 material was collected by Olive in Montgomery County, Indiana, who sent the material to Underwood, who, in turn, sent it to Ellis for identification. Ellis and Everhart published in 1895 a brief description of the causal organism, to which they gave the name *Phyllosticta solitaria*. The first report of blotch in commercial plantings was made by Clinton in 1902, at which time he called attention to the serious nature of the disease in southern Illinois. Clinton was also the first to study the cultural and morphological details of the causal fungus which he regarded as a new species of the genus *Phyllosticta*. After 1902 many workers reported blotch from apple-producing states in the eastern half of the United States. In 1907 Sheldon directed attention to the possible relationship of *Phyllosticta solitaria* E. & E. on the leaves of *Pyrus coronaria* L. to the disease on the leaves, branches, and fruit of *Pyrus malus* L. The relationship of the various infected organs of the commercial apple was established experimentally by Scott and Rorer, who were the first workers to conduct a general study of apple blotch. They studied the etiology of the disease and the cultural and morphological characters of the fungus and its life history and control.

Gardner emphasized the importance of petiole and bud scale infections as the origin of apple blotch cankers in 1922. In 1925 the monographic work of Guba cleared up many conflicting views concerning the life history and control of apple blotch. Kohl in 1932 added a number of important details, especially concerning the relation of weather to the infection periods, and confirmed the work of Gardner on the invasion of the twigs from the petiole infections. Very little has been added to our knowledge of the disease and its control since that time, although a few contributions have been made which have served either to emphasize the importance of earlier recommendations or to broaden our understanding of outstanding details.

Geographic Distribution. Since the first reports of *Phyllosticta solitaria* from Indiana and the vicinity of Washington, D.C., the disease has been identified from most of the states in the eastern half of the United States. It has been reported as far west as Kansas, as far north as Wisconsin, New York, and Maine, and to the coastal states on the East and South. It is not generally of commercial importance, however, north of the 40th degree latitude. As yet blotch has not been reported outside of the United States.

Economic Importance. In the apple-growing regions of the eastern half of the United States apple blotch is second in importance only to apple scab. In sections of this area where susceptible varieties and climatic conditions favor apple blotch, it is often the most important disease. In recent years blotch has decreased in importance largely because of the elimination of susceptible varieties such as Oldenburg (Duchess) and to more effective control measures. Loss of marketable fruit is extensive because of the unsightly blotches. On susceptible varieties where control measures are inadequate, this often amounts to 50 to 60 per cent of the harvested fruit. Cracking followed by rotting results from early infections. Stunting of growth and actual killing of fruit spurs are probably the most serious aspects of the disease. Apples on trees where spur injury is serious are always much smaller than on healthy trees even when no blotch occurs on the fruit. Petiole infections often result in extensive defoliation and consequent reduction in growth and fruit set. Fruit-stem infections cause dropping or dwarfing of fruit.

Symptoms. Blotch may cause serious injury to leaves, buds, twigs, and branches but is of particular commercial concern because of its effect upon the fruit of susceptible varieties.

Leaves. There are two distinct types of blotch lesions produced upon the leaves. The infections on the interveinal tissues of the leaf blade, which appear as circular yellowish-green spots about the size of pinheads, are of least importance. Shortly after the appearance of the spots a small black speck may be seen in each lesion (Fig. 10). In the early part of the season the leaf spots average about 1⁄16 in. in diameter and are so nearly the color of a normal leaf that they are often overlooked. As the season progresses, however, they become much more conspicuous, their light green color changing slowly through a brownish tan to a paper white. Leaf spots may vary from a few to over a hundred per leaf.

The more important type of leaf infection occurs on the veins and midrib on the undersurface of the leaves and on leaf petioles. These infections appear as elongated, sunken, light tan, or buff-colored lesions which produce a few small black specks (pycnidia). The lesions may vary in size from mere spots to nearly an inch in length. In the event of severe infection, they may girdle infected petioles, causing considerable defoliation.

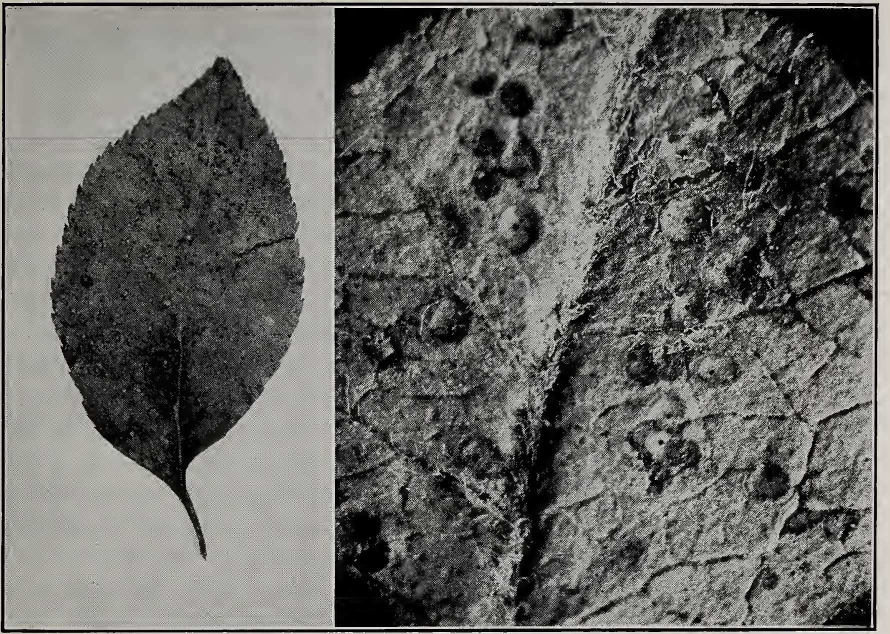

Fig. 10. Apple blotch on leaf, natural size and enlarged, showing single pycnidium in each spot.

Twig and Branch Cankers. The majority of blotch cankers on twigs and branches are located at leaf nodes or at the base of spurs that have developed from dormant buds, but a relatively small number occur internodally. New twig infections are not usually evident until late in the season, at which time they appear as raised dark purple to olivaceous spots. The size and shape of the lesion depend partly upon the time of infection and partly upon the nature of infected tissue. Lesions in firm, slow-growing twigs are usually much smaller and rounder than those in succulent twigs or water sprouts. Those in succulent tissue are definitely oval to elongate. Also, the more susceptible the variety, the larger the lesions are likely to become. New infections occur only on the current-year wood.

At the end of the first growing season small lesions usually remain purple to olivaceous in color, while the central portion of larger ones often becomes tan or light brown. Small black epidermal protrusions appear late in the season scattered rather evenly over the cankers. In many cases they do not actually break through the host epidermis until fungus growth is resumed the following spring. By spring the young lesions vary considerably in appearance. Most generally they are dried out and slightly sunken, although in many cases they actually protrude above the level of the surrounding healthy tissue. The lesion is often outlined by a small crack which separates the healthy and diseased tissues. Very early in the spring the vegetative growth of the fungus in the collenchyma cells of the cortex is resumed, causing a rapid increase in the size of the lesions. The tissues affected as a result of this growth appear dark olivaceous to purple in color and may be slightly raised. Small black dots appear in the newly affected tissue shortly after invasion is evident (Fig. 11). As a rule the portion of the lesion produced the previous season by this time has turned a light tan to orange tan in color. During the hot dry summer months the growth of the fungus is inhibited, but continues again for a relatively short period in the cool moist environment of early fall. Typical black specks appear in the fall-infected tissues late in the season. The original lesion continues this seasonal alternation of growth for many years, eventually causing large unsightly cankers which usually completely girdle a twig or branch. The older portions of the cankers have now become hard and dry, and much cracking and sloughing of infected tissue is evident. At this time the more recently invaded tissues form a

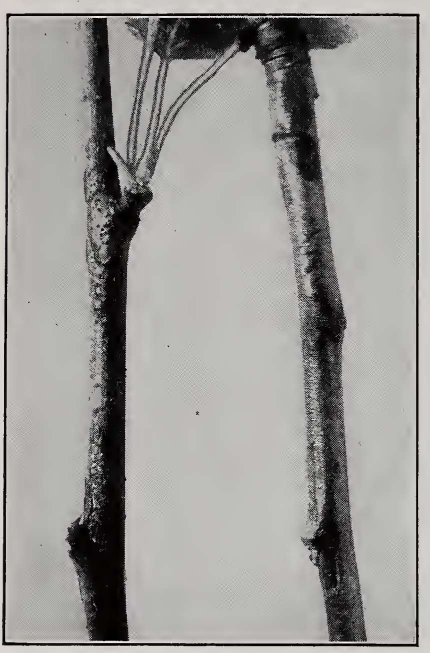

Fig. 11. Young blotch canker on twig, showing location at base of petiole and pycnidia on surface of lesion.

definite growth area around the margin of the canker. Under dead peeling bark the periderm layer develops to the extent of starving the fungus and repairing the wound. Cankered areas on older limbs are often spindle-shaped, owing to excessive growth of bark tissues.

Fruit. Fruit infections may occur practically any time from petal fall until harvest, depending largely upon the environmental conditions. Ordinarily initial infection occurs about 1 to 5 weeks after petal fall. Symptoms appear 2 to 3½ weeks after initial infection, depending to some extent upon weather conditions and the variety. The incubation period is shorter under conditions conducive to succulent growth and on the early-maturing varieties such as Oldenburg and Transparent. Fruit lesions appear as shiny black blotches varying from tiny spots to areas involving a large portion of the surface of the fruit. The larger areas usually represent the coalescing of several lesions and are especially common in damp seasons. As in the case of twig infections the lesions may be distinctly sunken or slightly raised. The edges of the lesions are irregularly lobed with many radiating outgrowths or projections (Fig. 12). The infected areas usually appear brownish black to black with small black dots or protuberances rather generally scattered over them. These black dots are the fruiting bodies of the fungus and are evident very shortly after the blotches themselves appear. As the blotches develop, the infected tissue becomes hard and dry, the growth of the underlying tissue is stunted, and the tension that arises from growth of the surrounding tissues results in the cracking of the apple across the lesion. This condition is especially noticeable at harvest on the later-maturing varieties.

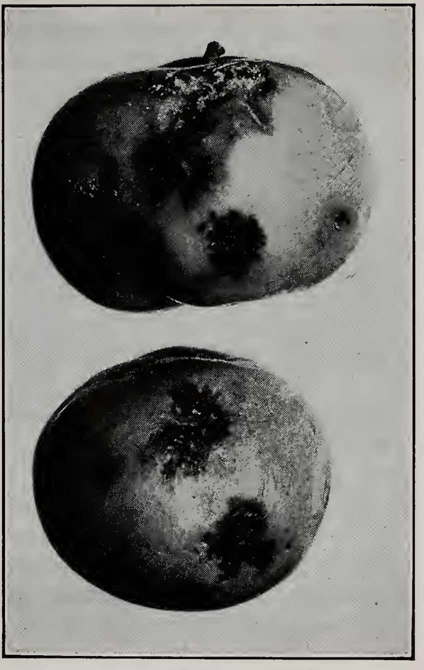

Fig. 12. Blotch lesions on apple fruit.

Causal Organism. The fungus causing apple blotch belongs to the Sphaeropsidales of the Fungi Imperfecti. No perfect stage has yet been found. It was first described by Ellis and Everhart as *Phyllosticta solitaria*[1] in 1895.

[1] Class Fungi Imperfecti, Order Sphaeropsidales, Family Sphaeroidaceae.

Hosts. Apple blotch is practically confined to certain species of the genus *Pyrus*, although Seaver has named *Crataegus* sp. (Hawthorn) as a host of the fungus. The cultivated apple, *Pyrus malus* L., is the most important host plant, although several species of crab apples are also seriously affected. The susceptible species listed by various authors are *Pyrus angustifolia* Ait (southern crab), *P. coronaria* L. (Garland crab), *P. baccata* L. (Siberian crab), and *P. lancifolia* Rehd. Apparently *P. ioensis* Bailey (common prairie crab) is entirely resistant.

Morphology and Cultural Characters. The *mycelium* of the apple blotch fungus in culture is septate with irregularly branched pale green hyphae. In young hyphae septa are rare and the walls are not constricted. In old mycelium the cells are short and thick and contain large globules of a greenish substance; septation is frequent, and the walls are prominently constricted at the septa and at the bases of the short, stout branches that arise irregularly. The uniting of these cells produces a very irregular network. Very little is known of the mycelium as it occurs within the host tissue, except that it is intercellular and practically confined to collenchyma tissue.

The *pycnidia* vary considerably in size and shape, the smallest occurring on the leaf spots. They vary in diameter from 60 to 95 μ, and each has a small rostrate ostiole measuring 9 to 12 μ long and 7 to 12 μ wide. The pycnidial wall is thicker at the top, and after the formation of the ostiole the thickened wall is retained around the short neck. A narrow sporogenous layer of hyaline parenchyma cells, bearing conidiophores, lines the interior of the pycnidium.

The pycnidia on the petioles and veins are commonly larger than those on the leaves (62 to 119 μ in diameter) but are similar in structure. The neck, or beak, of the pycnidium is rather prominent. Within the narrow, dark-colored wall the large hyaline sporogenous cells with conidiophores appear in sharp contrast.

On the fruit the pycnidia appear as few to many black prominent dots on the infected area. They are separated but may coalesce to form large aggregates. They are decidedly depressed or elliptical and almost twice as wide as deep. The lateral walls are 14 to 16 μ thick, while the basal wall is much thinner. The ostiole is indefinite, without a neck, the wall around it being about the same thickness as the lateral walls. The fruit pycnidia vary considerably in size and shape, but usually measure from 57 to 95 μ deep and 107 to 166 μ wide. After the spores are discharged in the summer, the sporogenous tissue which lines the interior of the pycnidium is rejuvenated and eventually fills the entire cavity. In the meantime, the ostiole is closed by the growth of the cell wall. During the fall and winter months the interior is filled with pseudoparenchymatous tissue enclosed in a thick carbonaceous wall. Extensive enlargement of pycnidia occurs, often resulting in their ultimate fusion. These fruiting bodies with dense, thick, carbonaceous walls, enclosing a hyaline parenchyma context, have been appropriately named *pycnosclerotia* and are referred to as such throughout this discussion.

On the bark there are distinguishable two types of fruiting bodies: first, the pycnidia, which form and function in the same season; and second, the pycnosclerotia, which form late in summer and fall, overwinter in a dormant condition, and function the following spring. In their early development both forms resemble in general shape and structure the pycnidia which are formed upon the fruit. In the spring the overwintered pycnosclerotia are comparatively large, with thick, carbonaceous walls and without a definite opening. Sporulation begins in the center of pseudoparenchymatous content early in the spring, and the sporogenous layer, which is formed gradually, progresses outward. When spore formation is complete, only the dark wall cells remain. The pycnosclerotia usually measure 155 to 274 μ wide and 107 to 238 μ deep (Fig. 13).

The *pycnospores* are ovoid, or broad-elliptical, unicellular, hyaline, multiguttulate, with a smooth wall often partially covered at the broad end by an elongated, gelatinous appendage. The spores measure from 7 to 11 μ long and 6 to 8.5 μ wide. The *conidia* produced from the pycnosclerotia and those produced in culture media differ from those produced in pycnidia which function in the season they are formed, in that a gelatinous appendage appears as a thick cap over the broad end of the spore. It is hyaline and at first very long and narrow and broadens out at the basal end where it often envelops as much as half the spore.

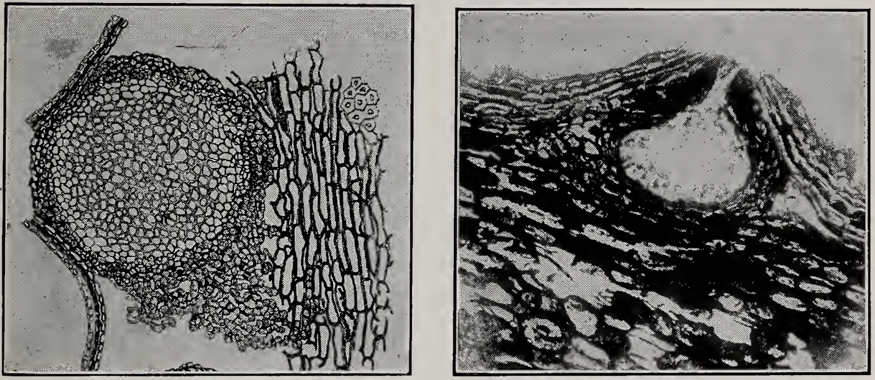

FIG. 13. Section of blotch pycnosclerotia from twig canker with central mass of cells which later round off as spores (left). At right, true pycnidium in petiole.

Disease Cycle. The apple blotch fungus overwinters as dormant mycelium in infected tissue. In this connection the cankers on twigs and branches are of greatest concern. Pycnospores are directly responsible for spring infections of petioles, leaf blades, fruit pedicels, and to some extent the current year's wood. Spring infections may occur as early as petal fall, although normally the greatest percentage occurs from 2 to 4 weeks later. The primary source of pycnospores is from the pycnosclerotia, which are confined to the older areas of the cankers. Irregular and continued exudation of spores from the pycnidia on the cankers are responsible for repeated primary infections throughout the spring and summer. In the fall, infections from these sources cease, for the supply of spores becomes exhausted and only pycnosclerotia are formed. The new cankers which appear in the fall likewise bear only pycnosclerotia which function the following spring.

Pycnidia are produced in great numbers on the spring infections of leaf petioles, blades, pedicels, and fruit, and account for the liberation of pycnospores as secondary inoculum. These pycnidia also become exhausted by early fall or sooner.

Twig and branch cankers result from the infection of current year's wood by two distinct modes. The most common is by the growth of the fungus from the collenchyma cells of infected petioles and pedicels through the undeveloped abscission layer into the collenchyma cells of the bark. The extension of the fungus into young twigs from infected dormant buds may also occur. The internodal type of twig infection usually results from direct germ tube invasion through the epidermis.

In the fall pycnosclerotia are produced on twig and branch cankers. With the advent of cold weather the fungus becomes dormant, in which condition it remains until weather favorable for growth occurs in the early spring. At this time the interior cells of the pycnosclerotia begin to differentiate and spores appear in the center. This process continues until the entire pseudoparenchyma context is differentiated. At about the same time that spores are being produced in the pycnosclerotia, the formation of new cankerous areas about new and old cankers is taking place. Fruiting bodies soon appear on this newly infected tissue, which give rise to pycnospores from middle to late spring. In late spring and fall pycnosclerotia are formed, which function the following spring.

The cycle described continues year after year, causing an annual enlargement of the cankers and an annual source of pycnospores for new infections. However, after about the third year the old cankers produce few pycnidia and probably account for very little infection. This is evidenced by the fact that when twig infection is controlled for several years, very little fruit infection occurs even when an inadequate spray program is followed.

Relation to Environment. Heavy rains and moderate temperatures are necessary for the exudation and subsequent germination of the spores of the apple blotch fungus. The pycnidia do not swell and exude their spores unless they are wet for some time. After the spores are discharged, several hours are required at favorable temperatures for their germination. The gelatinous appendage and the appressorium which is formed upon germination are capable of fastening spores to plant surfaces so thoroughly that driving rains do not dislodge them. From petal fall on, as long as moisture and temperature conditions are favorable, blotch infections may occur. Ordinarily, however, most infections take place in the few weeks following the second week after petal fall. Before this time temperatures are usually unfavorable for the rapid growth of the fungus or spore germination, and afterward the rains are not sufficient to permit spore discharge and subsequent germination. In general, it is agreed that blotch is a "warm wet-weather disease." Its environmental requirements undoubtedly account for its geographic distribution and severity in certain well-defined regions.

Control. Most of the research concerning apple blotch has had methods of control as its ultimate objective; consequently much has been written on this phase of the subject. The earliest recommendations for blotch control involved the use of Bordeaux mixture as a summer spray. Both ferbam and zineb have proved to be very effective in the control of apple blotch. When these are applied for several years, new twig cankers are eliminated and very little trouble is experienced in keeping the disease from attacking the fruit. The use of strong fungicides as delayed dormant sprays has been shown to have some value in reducing spring infections, but as yet such sprays in themselves cannot be relied upon to give satisfactory control. The use of varieties known to be moderately to highly resistant has met with general favor in areas where blotch is a serious problem. As a rule such varieties should not be planted near susceptible ones, since the fruit of most varieties is subject to infection even though the bark may be highly resistant to invasion. As in the case of apple scab, susceptibility and resistance seem to be relative, greatly influenced by general environmental conditions and, therefore, subject to local interpretation.

It has been demonstrated that canker eradication in nursery stock and young field plantings is a very effective method of controlling blotch. It may be accomplished by pruning or cutting out all visible cankers and spraying in the spring and summer to prevent the infection of current-year wood from any cankers which may have been overlooked. Canker eradication is especially applicable to young trees but unless followed faithfully is likely to be of no avail in the long run. Nursery stock should be carefully inspected and rejected if infections are evident.

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RUSTS OF POME FRUITS

Four rusts occur on cultivated pome fruits in North America. All of these are caused by species of *Gymnosporangium,* and all have an alternate stage on junipers (cedars). Three occur east of the Rocky Mountains, while the fourth is known only from the Pacific northwest (see Table 1, page 60 for differential characters). Since the disease caused by *Gymnosporangium juniperi-virginianae,* commonly known as "apple rust," is far more abundant than any of the others, it will be described in detail.

Apple Rust

Apple rust is also called "cedar rust" and "cedar apple rust." The name apple rust is appropriate since the apple is the only pomaceous host, while the other rust fungi attack other pome species as well as the apple.

History. Apple rust is of American origin and is not known to occur in any other country. It was described by Schweinitz on red cedar as early as 1822 and on wild crab apples a few years later, although at that time the connection between the two was not known and the latter was described as a separate species. In 1889, Thaxter first proved that the cedar gall fungus was the same as the one causing apple rust. Since that time a number of investigators have made successful cross inoculations.

In horticultural literature, few references are to be found to this disease before 1880. It would appear that no serious consideration was given to it until the growing of large commercial orchards of susceptible varieties called sharp attention to the possibilities of loss. Between 1887 and 1900 a number of experiments on control were undertaken, but no serious effort was made by commercial orchardists to include spraying for control of this disease in their spray schedules. In 1893, Jones, in Vermont, first demonstrated that removal of red cedars in the neighborhood of an orchard would give effective control. Between 1900 and 1920 attention was focused on this disease by a series of destructive epidemics in the eastern United States, especially in Virginia, West Virginia, and Pennsylvania. The studies by Giddings and his associates in West Virginia cleared up a number of problems in connection with the time of infection and seasonal susceptibility of the host. Heald in 1909 reported on the life history of the apple rust fungus and discovered the biennial character of the stage on the red cedar.

A clearer understanding of the characteristics of the three rusts of apple in eastern United States was obtained as a result of the investigations of Weimer (1917), Thomas and Mills (1929), Miller (1936, 1939), and Bliss (1933).

Geographic Distribution. In North America apple rust occurs from Maine to North Dakota and from southern Canada to Florida and Texas. It is not known to occur in the Pacific Coast section but has been reported from Alaska. It is most destructive in the Middle Atlantic states, especially in the mountainous sections of Virginia and West Virginia. Severe epidemics also occur in the Mississippi Valley states. In Iowa and Nebraska where red cedar windbreaks were commonly planted for the protection of the orchards, this disease has taken heavy toll in favorable seasons.

Economic Importance. Losses from apple cedar rust are due to three conditions: (1) fruit infection and consequent loss of grade, (2) reduction in size and quality of fruit as a direct result of defoliation, and (3) devitalizing of tree and consequent reduction in yield and size in subsequent years. In some seasons severe fruit infection may reduce the crop as much as 10 to 50 per cent in individual orchards. Early-leaf drop results when 10 or more lesions occur on a leaf and the resulting defoliation reduces the size and quality of the fruit. Trees devitalized by the rust often fail to set fruit the following season, and thus the loss may be extended over a period of several years. Trees suffering from infection over a series of years are stunted and fail to produce fruit.

Symptoms. Rust occurs on leaves, twigs, and fruit. On the *leaves* the small pale yellow spots appear on the upper surface in May. These gradually enlarge and turn to an orange color. The tissue of the leaf is not killed but is somewhat swollen (hypertrophied). On some varieties the tissue dies early and brown necrotic areas result, but this is not common. When the spots attain a diameter of about ⅛ in., orange-colored drops of exudate may be seen near the center of the spot, and later black dots appear on the upper surface (Fig. 14). At this time the spot is also evident on the undersurface, but the color remains yellow rather than orange. The final size of the lesions varies with the variety and the number of spots on a leaf. As many as 300 spots have been reported as occurring on a single leaf.

The diseased tissues become decidedly thickened on the undersurface and in July and August a number of protuberances appear, which later develop into open, cylindrical tubes. The distal end of these tubes is split in segments which are curled back on themselves (Fig. 17). In these tubes may be seen light brown spores. The tissue of the leaf may remain alive through the greater part of the season, but loss of vitality often results in the drop of leaves. Infected leaves are also smaller than normal ones. Defoliation is especially pronounced under dry summer conditions.

On the *twig* the disease appears only on the current season's growth.

The area involved is usually not more than an inch in length and consists of a swollen section of the twig on which are developed the characteristic cylindrical fruiting bodies.

On the *fruit* the lesions are usually found near the calyx end. At first spots appear similar to those on the leaves but are usually much larger. The normal light-green color of the young apple changes to a darker green around the border of the orange-colored infected area (Fig. 15). Often the cylindrical protuberances fail to appear, but when present they are usually in a circle outside the black dots, or pycnia. These fruiting bodies are borne on a raised roughened cushion of tissue, but often the whole fruit is misshapen as a result of the attack of the fungus. A cut into the flesh below the spot will show that the tissue is still alive, although somewhat corky. This is in sharp contrast to the necrotic area which is found in the case of apples attacked by the quince rust fungus.

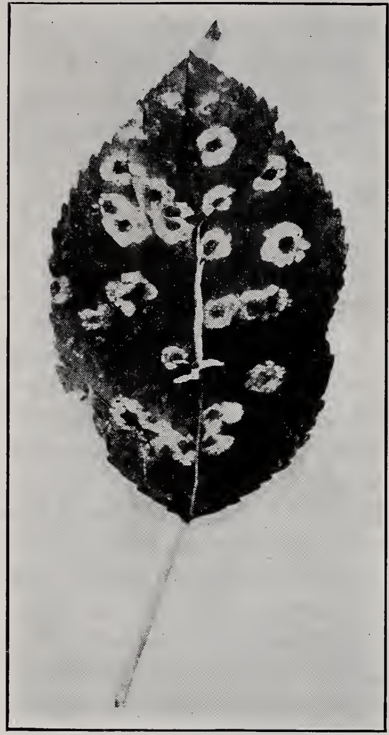

Fig. 14. Apple rust on upper leaf surface showing central dark pycnia, and outer orange zone (white in figure).

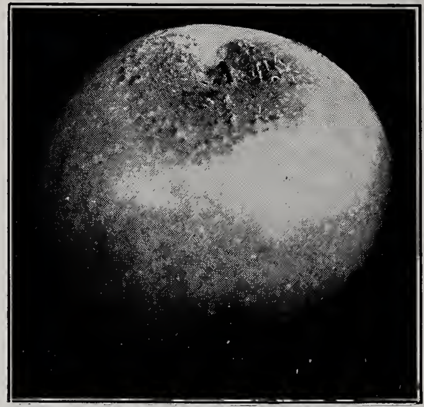

Fig. 15. Apple rust on fruit. A few tubular aecia at right, immature aecia below.

On the *cedar* the apple rust fungus produces a deep-brick-red to chocolate-brown gall which may vary in size from that of a grain of wheat to 2 in. in diameter. These are frequently referred to as "cedar apples." In shape they are usually globular but may be reniform. The new galls which are to produce spore horns the following spring are first seen in June as small deep-red globular structures in the axils of the leaves (spines). At first smooth, they later show a series of circular depressions over the surface. In the center of each of these depressions may be seen a small pimple-like protuberance. The flesh of the gall is corky in texture.

Early in the spring following rainy periods, a gelatinous yellow spore horn from ¼ to ½ in. long and about ⅛ in. in diameter pushes out from the center of each of the circular depressions on the gall (Fig. 16). When wet these extend as much as 2 in. from the gall. During dry weather the horns contract into thin, wrinkled threads, but when moistened they swell again to their original size. Later in the season they drop off, and the gall turns black and dries out. These old blackened galls may remain on the tree for several years, but do not function again. The number of spore horns produced on a single gall varies from one to over a hundred. Heavily infected cedar trees covered with the expanded galls are very conspicuous from a distance.

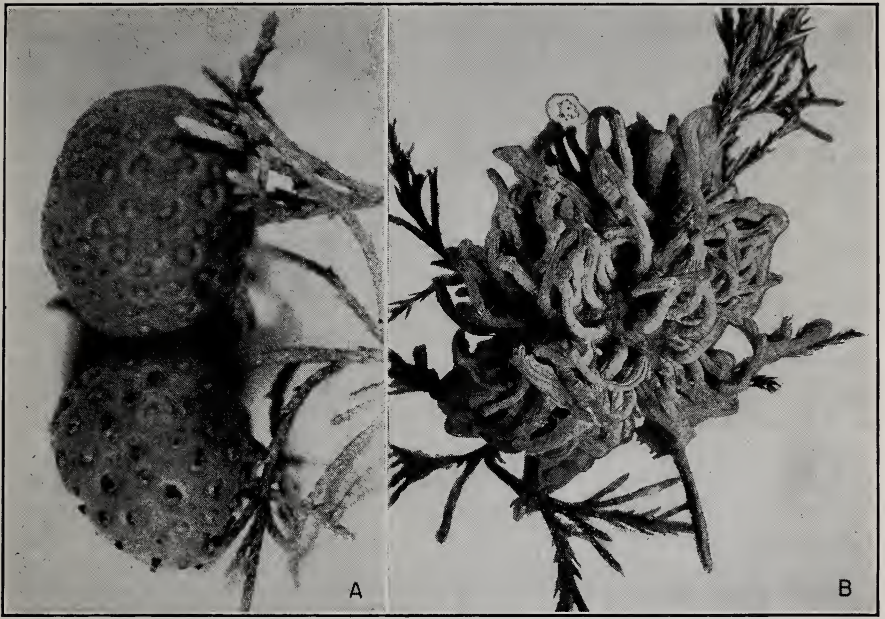

Fig. 16. Dormant condition of cedar apple showing pits from which spore horns arise (left). At right, expanding spore horns.

Causal Organism.[1] Apple rust is caused by one of the true rust fungi, *Gymnosporangium juniperi-virginianae* Schw. Fortunately, the nomenclature of this fungus has not been confused by numerous synonyms. The stage on the apple was first described by Schweinitz in 1822 as *Caeoma* (Aecidium) *pyratum*, which was later changed by Thaxter to *Roestelia pyrata*, under which name it was known until the connection with the

[1] Class Basidiomycetes, Order Uredinales, Family Pucciniaceae.

cedar fungus was made. Link, in 1825, called the rust *G. macropus*, and this name is often seen in older literature.

Hosts. The *Roestelia* aecial stage occurs only on species of the crab apple and cultivated apple. According to Crowell, "all of the ornamental crab apples of American origin are susceptible, and all of the Eurasian apples, except the highly resistant *Malus sylvestris* and possibly *M. floribunda*, are immune." On junipers the telial stage has been reported on *Juniperus virginiana*, *J. scropulorum*, and *J. horizontalis*. All other species of juniper seem to be immune.

Morphology. The galls formed on the cedar are the result of foliar infection by the aeciospores from apple. The galls themselves are composed of the hypertrophied leaf tissue in which is present the mycelium of the fungus. The hyphae are sparsely septate and average about 2.5 μ in diameter. Haustoria are present but by no means abundant. Telial horns arise from aggregates of mycelium in the galls, which first appear as early as October, the year preceding their extrusion. The young horns show immature spores by December. The circular depressions described under symptoms are the result of retarded growth of the gall tissue in the region where the spore-horn initials are developing (Fig. 16). The spore horns are composed of a great number of teliospores borne on elongated stalks. The growth of the spore horns ruptures the epidermis of the gall, and on wetting, extensive gelatination takes place, with the result that the horns are rapidly expanded. The horns of this species are cylindric in shape, golden brown to yellowish in color.

The *teliospores* are two-celled (occasionally one- or three-celled), 15 to 22 by 33 to 65 μ, narrowly ellipsoidal to rhombic-oval in shape, not constricted at the septum. There are two germ pores in each cell, one on each side near the septum. The *promycelium* (basidium) is four-celled and typically bears four hyaline *sporidia* (basidiospores), on short pointed projections, *sterigmata,* formed near the upper end of the cells.

The *pycnia* are flask-shaped structures produced on the upper surface of the leaf spot. They develop from a mass of tissue under the epidermis. The one-celled, uninucleate, ellipsoid, hyaline *pycniospores* are borne on elongated sporophores. The spores are from 3 to 4 μ in diameter. A clear honeylike mass of material, golden yellow in color, is forced out of the pycnia along with the spores.

The *aecia* (Fig. 17) are produced on the lower surface of the lesion, but they originate deep in the leaf tissue and extend downward through the hypertrophied cushion. The cylindrical aecium has an outer wall, the peridium, which is a single layer of cells in thickness. Arising from a mass of cells at the base are the short, club-shaped sporophores on which the aeciospores are borne in long chains. The *aeciospores* are binucleate and measure 16 to 24 by 21 to 31 μ. The peridial wall extends some distance beyond the surface of the leaf, but finally splits into narrow strips which become recurved during dry weather. Pycnia and aecia are also produced on the fruit.

Disease Cycle (Fig. 18). Extrusion of the spore horns begins during March, April, or May, depending upon the latitude. After breaking through the surface they may protrude for ¼ in. or more (Fig. 16). When rains soak the galls, the spore horns become gelatinized and swell very rapidly. The stalks of the teliospores elongate, and the spores near the surface of the horns germinate within 4 or 5 hours, each producing eight sporidia (four to each cell). It has been estimated that a single large gall will produce as many as 7,440,000,000 sporidia. As the spore horns dry, the sporidia are forcibly ejected from the sterigmata on which they are borne. These either drop from the surface of the horns or are carried by wind currents and are thus dispersed to the developing apple leaves in the neighborhood of the cedars. The sporidia are carried by wind to great distances, but if allowed to dry, soon lose their vitality. The distance which they travel and still retain their ability to infect has an important bearing on the practicability of cedar tree removal in an orchard section. While several billion sporidia may be produced and discharged from a single

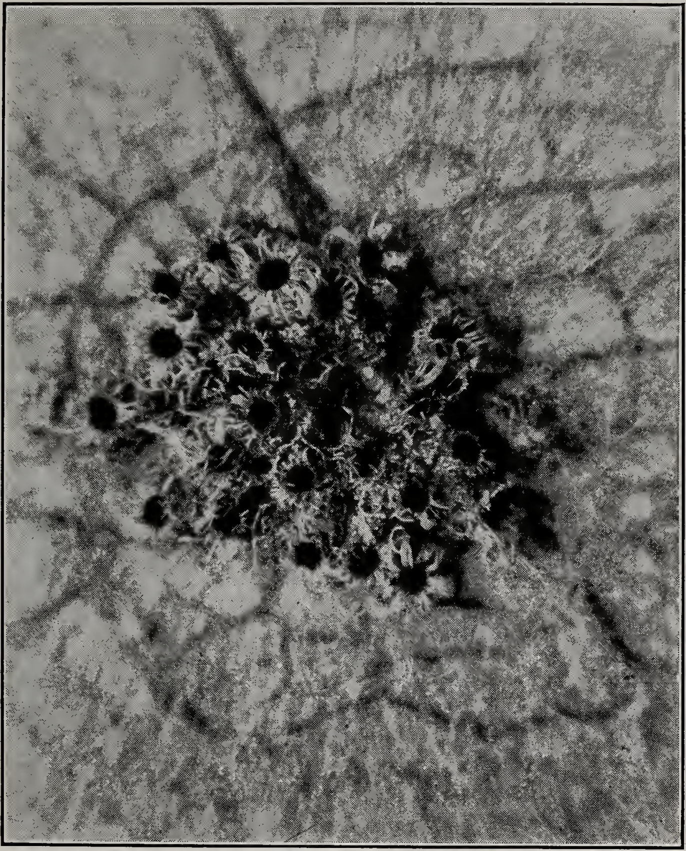

Fig. 17. Apple rust aecia on leaf, enlarged to show curved segments of peridium and central mass of dark aeciospores.

small cedar tree, infection of apples is not general beyond a radius of 4 or 5 miles from the source of sporidia production. It would seem, therefore, that either the sporidia are not carried far or that they quickly lose their vitality. Since there is no reason why spores of this character should not be carried many miles by strong currents of air, it may logically be deduced that loss of vitality is the true explanation of the limited range of infection.

Infection takes place through direct penetration of the cuticle on the upper surface of the leaf. The incubation period is about 10 days, but often no evidence of the disease can be seen for 12 to 14 days after infection. Infection may occur within 5 or 6 hours after inoculation. Thus a rainy period of a few hours will suffice to germinate the teliospores, while if this is followed by a short dry period, discharge and dissemination of the sporidia will occur and another short period of rain will bring about infection. It has been proved by Giddings and others that the leaves of the apple are susceptible over a comparatively short period in their development.

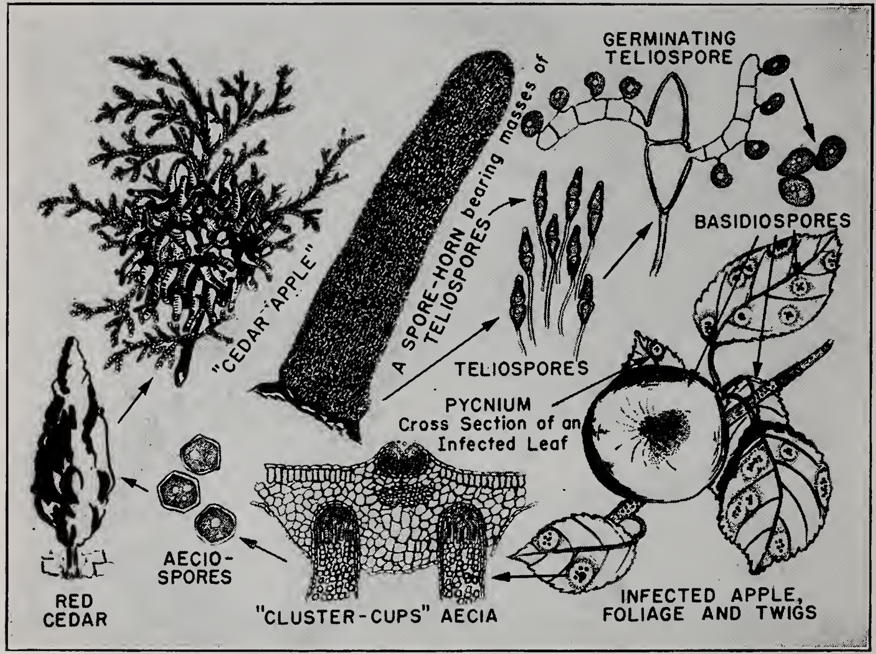
GERMINATING TELIOSPORE
A SPORE-HORN bearing masses of TELIOSPORES
BASIDIOSPORES
"CEDAR APPLE"
TELIOSPORES
PYCNIUM
Cross Section of an Infected Leaf
RED CEDAR
AECIO-SPORES
"CLUSTER-CUPS" AECIA
INFECTED APPLE, FOLIAGE AND TWIGS

Fig. 18. Life cycle of the apple rust fungus.

Most of the infection takes place during April, May, and June, after which the telial horns are exhausted, and in addition most of the leaves are immune. Pycnia are produced shortly after the spots become plainly visible. Until recently the function of the pycnia and pycniospores was not known. It has been demonstrated by Miller that the mixing of the exudate from different pycnia is essential for the production of aecia and aeciospores. It is a matter of common observation that many of the rust spots on the apple are sterile so far as the production of aecia are concerned. This condition may be explained on the above basis. This phenomenon is called *heterothallism* and is now regarded as of common occurrence in rust fungi.

The aecia do not develop until several weeks after the appearance of the pycnia. The cushion of hypertrophied tissue on the undersurface continues to enlarge until some time in July, when there is evidence of the aecia, which appear as brown papillae scattered over the surface of the cushion. The aeciospores on drying fall out of the cylindrical tube in which they are borne. The old spores are continuously pushed out by the developing young spores beneath. As the aeciospores drop from the aecial tube, they are carried by air currents to any cedar trees in the neighborhood. Germination and leaf infection follow under favorable conditions. It appears that the aeciospores germinate best after being aged or matured and that more will germinate in the fall months than earlier. The optimum temperature for aeciospore germination is 75°F, which would indicate better conditions for infection during the warmer months of late summer and early fall. Weimer is of the opinion that infection of the cedar takes place in September or October, while Miller and others suggest the possibility of the spores remaining dormant in the leaf axils until spring, when infection might occur on the young leaves. Heald successfully controlled infection on cedars by spraying in July and August, thus indicating that late-summer infection may occur.

On the cedar, regardless of whether infection takes place in the fall or early spring, the galls do not appear until the following June, when they may become evident as small globular bodies in the axils of the leaves. These galls are not destined to produce spores until the following spring, so that if infection of the cedars takes place in September of 1952, the spore horns are not produced until April of 1954, about 19 months later; thus there is a 2-year life cycle.

Relation to Environment. Because of the complex life cycle and the heteroecious nature of the apple rust fungus, its relation to enviroment is of more than usual importance. The fact that apple rust did not assume great commercial importance earlier in the history of apple growing in the United States may be explained on the basis of the fact that only a special sequence of favorable conditions can bring about a widespread epidemic. It is probable that the rust fungus in its earlier history was confined to areas where the crab apple and the cedars were in close proximity, a condition which prevailed especially in the southern

Appalachian region. In Virginia and West Virginia rust epidemics started when a very susceptible variety, York Imperial, was planted extensively in the regions where the cedar trees were abundant.

As pointed out by Giddings, infections are conditioned by the following: (1) development of rust galls on the cedar, (2) meteorological conditions, (3) development of apple foliage, and (4) location of the orchard with relation to the cedar. In the spring the swelling of the spore horns, germination of teliospores, dissemination of sporidia, and infection of the apple are dependent upon a sequence of meteorological conditions. Wet weather is necessary for the gelatination of the spore horns and subsequent germination of the teliospores. This process, however, takes place with remarkable rapidity, since it has been shown that from 4 to 6 hours after wetting the whole process may be completed. The spore horns after germination of the teliospores must dry somewhat to effect the discharge of the sporidia and their dissemination. Wind currents sufficient to carry the sporidia to the apple are necessary. Thus Giddings records one discharge of sporidia sufficient to show a yellow coating on a white card placed beneath rust galls for 2 hours, and yet no infection occurred on apples a few rods away. This he explains on the basis of lack of wind, since other conditions were favorable. Optimum temperature for natural infection of the apple is around 65°F. At 55°F little natural infection takes place, while at 50°F no sporidia are discharged. The leaves become immune to apple rust in from 18 to 28 days after they unfold, depending on conditions of growth.

The distance between orchards and cedars in relation to degree of infection is dependent upon topography and direction and velocity of the wind. An orchard at the base of a hill on which cedars are growing may be more severely infected than one an equal distance from cedars in a level area. Likewise, orchards located in the line of the prevailing winds in relation to infected cedars may be severely infected, while those in the opposite direction may escape entirely.

Very dry summer weather frequently results in the absence of galls on the cedars the following year. Thus the extensive drought of 1934 in the Mississippi Valley resulted in an almost total absence of cedar galls in 1935 and a consequent lack of apple rust in the spring of 1936. But in southern Illinois, where occasional rains occurred in 1934, cedar galls were common in 1936 and a moderate infection of rust of apple occurred.

Control. Methods of control of cedar rust are indicated by the life history of the fungus. As early as 1893, Jones gave concrete evidence of the value of red cedar eradication in the neighborhood of an orchard. Since that time extensive cedar eradication has been undertaken in several sections of Virginia and West Virginia. These eradication programs have proved successful in reducing the amount of rust to a point where it is of little commercial importance when most of the cedars within 2 miles of the orchards have been removed. In general, the removal of cedars is practical under conditions where the cedars themselves are of no particular value or where they are few in number. Red cedars should not be planted for windbreaks for orchards. Cutting out of wild crab trees in the area between cedars and the apple orchard to be protected as well as from among the cedars is advisable, since the crab apple may serve as a "bridging" host for continuous reinfection of cedars and thus increase the inoculum for apple infection.

In ornamental plantings it is sometimes advisable to remove the galls from the cedars during the winter previous to the formation of spore horns (second winter after infection). Some of the smaller galls may be overlooked, but the chances of apple infection will be materially reduced by this practice. Rust-resistant selections of the red cedar have been found and may prove of value for ornamental plantings.

Next in importance to the removal of cedars in the control of rust is the selection of rust-resistant varieties of apples. Some confusion has resulted from attempts to compile lists of resistant and susceptible varieties by investigators in various sections of the country. This has been due in part to limited observations and possibly to variations or strains in the rust organism. On the whole, however, it has been well established that certain varieties are highly susceptible under all conditions, while others are decidedly resistant, or possibly entirely immune.

Crowell in 1934 compiled a very complete list, giving the relative susceptibility of a large number of varieties. This list shows that of the common varieties the following are regarded generally as susceptible: Jonathan, Red June, Rome, Wealthy, York Imperial; as slightly susceptible or immune: Arkansas Black, Baldwin, Delicious, Early Harvest, Maiden Blush, McIntosh, Oldenburg, Northwestern Greening, Red Astrachan, Stayman, Wolf River, Transparent. Varieties such as Ben Davis, Gano, Grimes, and Golden Delicious are in the doubtful list, but may be safely planted where rust is not especially prevalent on susceptible varieties. It is evident that in a region where the red cedar is abundant, it would be very unwise to plant varieties such as Wealthy and York Imperial, known to be highly susceptible to the disease.

Until about 1940, spraying for the control of apple rusts was considered unreliable mainly because of the necessity of spraying at frequent intervals during the critical period of infection. Ferbam is very efficient in the control of apple rusts. Applications of it are made in the "pink," calyx, and first and second cover sprays. If the blooming period is prolonged, a full-bloom spray is advisable for protection of the rapidly expanding foliage.

Sodium cresylate has been used on junipers to kill the developing spore horns on the galls. This treatment must be timed to coincide with the emergence of the spore horns from the galls in early spring. This method is rarely used because of danger of injury to the cedars. Protection of cedars of special value may be obtained by spraying them with ferbam during the late summer, when dispersion of the aeciospores from the apple takes place, that is, during late July and August.

TABLE 1. DIFFERENTIAL CHARACTERS OF APPLE RUST FUNGI

Name	On cedar	On apple leaves	On apple fruit	Microscopic
Gymnosporangium juniperi-virginianae (apple rust)	Forms large regular galls on leaves (needles); spore horns cylindric; repeated sporulation; biennial	Similar to *G. globusum* on upper surface; aecia numerous, scattered; peridia revolute, regular	Orange-colored, usually raised; no necrosis; aecia often present; pycnia abundant, open, black	Peridial cells long, narrow, strongly curved, separate in water
G. globosum (hawthorn rust)	Galls usually small on leaf; spore horns clavate; single sporulation; perennial (3–5 years)	Lesions small on apple (large on hawthorn); aecia few, closely aggregate in center of lesion; peridial tubes long; irregular dehiscence	Rare on apple fruit (very common on haws)	Peridial cells irregularly lanceolate, adhere in plates in water, only slightly curved
G. clavipes (quince rust)	Galls spindle-shaped enlargements of twigs; perennial	Not known on apple foliage	Dark green, sunken, or craterlike; necrotic areas deep in flesh of apple; no aecia; pycnia few and undeveloped or absent	Peridial cells in plates, verrucose on outer walls; aeciospores larger than others

Quince Rust

Next in importance to apple rust is quince rust, so called because until recently it was considered primarily a disease of quince. Within recent years its importance on apples has been realized, mainly as a result of a better understanding of the character of the symptoms of this rust on the apple fruit.

History and Geographic Distribution. The fungus causing quince rust is undoubtedly of American origin. It was first described by Schweinitz in 1832 and is not known to occur outside of North America. In this country it ranges from Newfoundland to British Columbia and southward east of the Rocky Mountains to northern Florida and Texas and into Mexico.

Economic Importance. Losses from quince rust are not extensive on the quince, since this fruit is of relatively little economic importance in North America. However, on quinces that are grown in regions where the red cedar occurs, the fruit may be attacked year after year and is rendered worthless.

The main economic loss is to the apple. Since the symptoms of this rust on apple fruit have been clearly recognized, the loss on susceptible varieties such as Red Delicious has been estimated at 1 or 2 per cent in epidemic years. On the whole, however, the disease is much less important than apple rust since there is no damage to the foliage.

Ornamental hawthorns are often rendered unsightly by the attack of this rust on fruit and stems.

Symptoms. On quince the fungus rarely attacks the leaves and, if present, is confined to the veins, which become swollen and thus cause curling of the leaf. On the *fruit*, the disease is very conspicuous when the long, white aecial tubes appear. These may cover the whole fruit or occur on one side only. Terminal buds are attacked, and the twigs die at the end of the season.

On *Crataegus* (hawthorn) the symptoms on the haw fruits are similar to those on quince.

On *apple* fruit the disease is rather difficult to detect on account of the fact that there is little evidence of pycnial or aecial development. Usually the diseased fruit becomes puckered at the blossom end when it is about an inch or less in diameter. The surface of the lesion is dark green in color. As the fruit enlarges, this end of the apple remains constricted (Fig. 19). Unlike apple rust, the flesh below the lesion shows a dead area often extending to the calyx tube. The dead tissue is brown and spongy. In some cases there is evidence of aecia, but these rarely develop and produce spores.

On the *cedar* the galls are found as spindle-shaped enlargements on the stems. These fusiform swellings are hard to detect in the early stage, but since the fungus is perennial in the galls, the swellings become more conspicuous as the hemispherical orange-brown telial sori develop over the surface. In damp weather these gelatinize and become conspicuous even at a distance.

Causal Organism. Quince rust is caused by *Gymnosporangium clavipes* Ck. and Pk. It was first collected and named *Caeoma germinale* by

Schweinitz in 1832. It was described in some detail by Farlow in 1880. The differential characters of this rust are given in Table 1, page 60.

The cylindrical *aecia* are crowded on the fruit (quince and hawthorn) with conspicuous white peridial walls. Aeciospores are globoid 21 to 32 by 24 to 39 μ with pale yellow walls coarsely verrucose. Uredia are wanting. The *teliospores* are ellipsoid, 18 to 26 by 35 to 51 μ with yellowish walls.

Hosts. The telial stage occurs on *Juniperus communis*, *J. sibirica*, and *J. virginiana*. The aecial stage is found on a number of species of *Amelanchier* (juneberry, etc.), *Aronia* (chokeberry), and *Crataegus* (hawthorn), as well as on quince and apple.

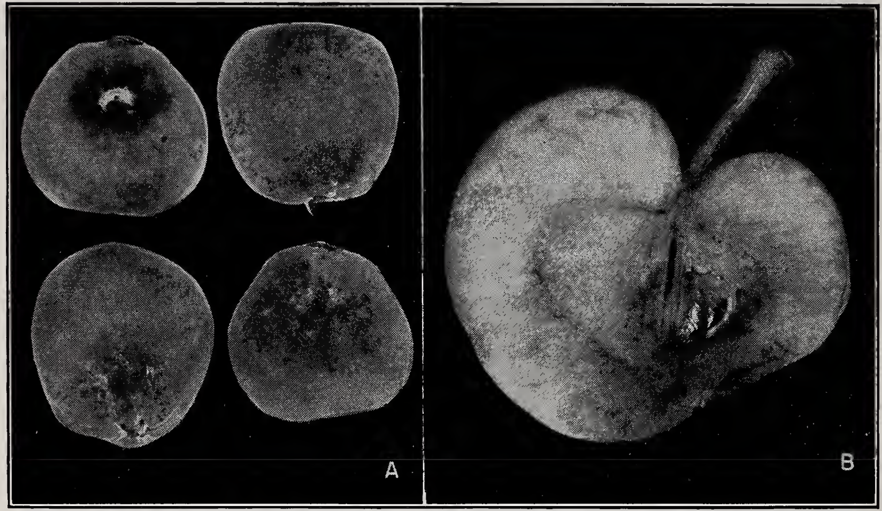

Fig. 19. Quince rust on apple fruit showing puckered external appearance (left) and deep-seated browning (right).

Disease Cycle. Since the uredinial stage is wanting, infection on the pome fruits must come from the basidiospores produced by the teliospores on the junipers. However, this rust differs from apple rust in that it is perennial in the lesions on the juniper, and thus once infected, the galls remain a source of inoculum over a period of years. The teliospores germinate upon the gelatination of the telial sori and the basidiospores are carried by air currents to neighboring orchards or to wild hosts, especially the hawthorn. Apple fruit infection usually occurs fairly early in the spring; that is, shortly after petal fall as is the case with apple rust. Usually infection on the apple fruit is confined to the calyx end but occasionally may be on the side. On quince, hawthorn, and juneberry it is usually more generally distributed.

Inoculation of junipers is from the aeciospores produced on quince, wild hawthorns, or juneberries, since few, if any, aecia are produced on apples. Since these wild hosts are very common in regions where junipers occur, a source of infection is usually present.

Varietal Susceptibility. Varieties of apple susceptible to apple rust are often resistant to quince rust. For example, Miller in 1932 in a survey of orchards in southern Indiana found quince rust on fruits of Winter Banana, Winesap, Baldwin, Stayman, Delicious, and King David, while Jonathan, Rome, Ben Davis, and Wealthy, which are very susceptible to apple rust, were free of quince rust. In Illinois quince rust is much more common on Red Delicious than on any other variety.

Control. Destruction of cedars in the neighborhood of apple orchards is imperative in eliminating the source of infection.

Sprays effective against apple rust have proved satisfactory for the control of quince rust on apple.

Hawthorn Rust

Hawthorn rust caused by *Gymnosporangium globosum* Farl. is of minor importance on cultivated pome fruits. It may cause considerable foliage injury in some years on apple and pear, but does not affect the fruit. For differential characters use Table 1, page 60.

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Apple Bitter Rot

The common name "bitter rot" has been used as descriptive of this disease since it has been recognized as a specific malady of the apple. As early as 1850, Baker, reporting on the fruits of Morgan County, Ohio, speaks of one variety as "subject to the old 'bitter rot' (but) seldom attacked by the new disease bearing this name." Thus it is evident that old references to this specific disease under the common name should be regarded with suspicion. "Ripe rot" is occasionally used as the common name.

History. The disease bitter rot and the organism causing it were described by Berkeley in England as early as 1856. The bitter rot fungus was first definitely identified in America in 1867, but it is evident from a perusal of the old horticultural writings that it was well known and dreaded as a disease before this time. Coxe gave an accurate description of the disease as observed near Philadelphia in an unpublished manuscript evidently written before 1828. It is impossible to determine the original home of the bitter rot fungus, but since it occurs on a number of other hosts, it was probably present in some form in this country in early colonial times. As stated above, frequent references can be found to a disease called bitter rot by growers, but with few exceptions it is not possible to determine whether or not they are referring to this specific disease. Clinton in 1902 gave the first description of the perfect stage of the bitter rot fungus on apples. Burrill and Blair in 1902, and Spaulding and von Schrenk in 1903, described bitter rot cankers on apple wood and their relation to primary infection of the fruit. The discovery of bitter rot cankers is credited to Simpson of Vincennes, Indiana, who noticed the conelike infections of the fruit below blackened cankers on the branches. The primary cause of those cankers was not determined at the time.

A number of investigators have demonstrated by cultural and cross-inoculation studies that the bitter rot fungus is not confined to the apple and that many described species of the genus *Gloeosporium* from other hosts will bring about typical bitter rot symptoms on apple.

Geographic Distribution. Bitter rot is known to occur in practically all countries where the apple is grown commercially. This is to be expected in the light of the wide host range of the causal fungus. While it occurs in other countries, it is of major economic importance only in the United States, and in this country its worst ravages occur in a restricted area. This area may be roughly defined as extending from the Atlantic west to central Kansas and south from the 40° meridian to the southern limit of apple growing. It is not known to occur west of the Rockies. Although it has been reported from most of the states east of the Rockies, it is of minor importance where either cool or dry summer weather prevails.

Economic Importance. Bitter rot may cause enormous losses because of the fact that an entire crop of apples may be lost within a few weeks of harvest after the grower has gone to the expense of producing the crop. Fortunately, such severe losses occur only under exceptional weather conditions and are restricted to comparatively small areas. The loss is confined to the damage done the fruit in the orchard. On account of the high-temperature requirements for the development of the fungus, little loss is experienced in modern transit and storage.

Very heavy losses from bitter rot were common during certain periods between 1880 and 1910. The general improvement in spray practices and sanitation probably accounts for the general decline in the losses from the disease since the early part of the present century.

Symptoms. On the fruit, bitter rot may appear when the apple is half grown, but more frequently not until it approaches its full size. The rot starts as a small circular area, light brown in color. These spots enlarge rapidly, becoming definitely circular in outline and somewhat sunken in the center, thus forming a saucer-shaped depression. The number of spots on a fruit may vary from one to many. They bear no relation to insect injury or the lenticels, thus distinguishing them from black rot and Jonathan spot. Usually when a large number of spots occur on a single fruit, only a few develop into the advanced stage. Until the spots are about ½ in. in diameter, the surface remains smooth and may change in color to a darker brown or may turn almost black, depending apparently on the variety attacked and possibly to different strains of the fungus involved. After the spot attains a size varying from ½ to ¾ in., there appear a number of slightly raised cushions on the surface, usually most prominent near the center, but extending outward almost to the edge of the spot (Fig. 20). Usually, but not always, this is accompanied by a decided darkening of the tissue beneath the cushions. Under humid conditions the cushions develop quantities of spores in a gelatinous or creamy mass, salmon pink in color, and often arranged in concentric circles. The concentric arrangement is often pictured as typical of this disease, but an indefinite distribution of the cushions and masses of pink spores is as common as is the concentric arrangement. As the rotted area increases in size, other rings of pustules appear. The rapidity of the rot and of the formation of concentric rings or irregularly arranged pustules depends upon weather conditions, warm humid weather favoring a more rapid development of the rot and the formation of numerous rings outside the original central sporulating area. As the spots become older, the pink masses disappear and a dark brown to black, somewhat wrinkled and sunken spot results.

Beneath the exterior evidence of the spot certain changes are taking place in the flesh of the apple which need to be followed. Soon after the spot appears on the surface, a shallow rotted area may be found in the flesh beneath the spot. This progresses toward the core and spreads outward as the surface spot increases in diameter, until a cone-shaped area is involved with the apex of the cone at the core.

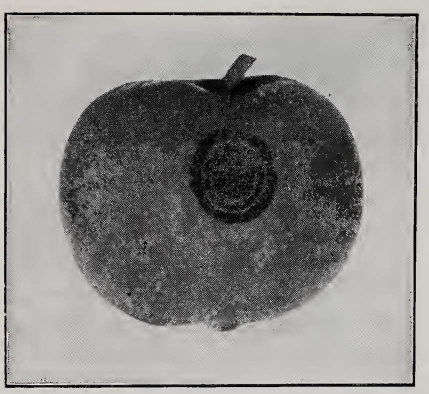

Fig. 20. Bitter rot on fruit showing concentric rings of acervuli and spore masses.

The rotted flesh is somewhat watery in character, but is not distinctly mushy as in the case of blue mold or leathery as in black rot. While the name bitter rot was given this disease, the flesh may or may not be bitter-tasting.

Usually more than one spot develops on a fruit, and by the fusion and extension of these spots the entire fruit is rotted. Premature ripening and dropping of the apples is common, but in some cases the mummied fruit remains attached to the twig throughout the winter. After falling the diseased apples do not entirely disintegrate but often mummify, although not to the extent found in black rotted fruit.

Bitter rot infections sometimes occur in the fall, just before harvest. The growth of these spots is checked by cool weather, and their appearance may be quite different from the rapidly growing spots described above. They are small red to purple lesions with a dark center, somewhat resembling Jonathan spot. They develop very slowly under cold storage, but upon removal from marketing they may develop very rapidly at room temperature.

Bitter rot *cankers* on the limbs, as explained later, appear to be developed only under exceptional conditions, since they are rarely encountered. Cankers are found on branches which are at least 2 years old. Roberts describes them as follows:

The bitter rot canker consists of a sunken portion of the bark, usually somewhat oval in outline, beneath which the wood is dead and dry. The dead bark and cambium adhere rather firmly to the wood, and in older cankers more or less complete cracks or fissures parallel to the edges of the cankers, giving a zoned effect to the dead bark. The wood and medullary rays, even in the case of young cankers, are colored brown all the way into the pith. Often the canker is surrounded by a layer of callus, which successfully prevents further extension of the canker, and which eventually entirely obliterates the old lesion.

Causal Organism. The fungus causing apple bitter rot is now generally called *Glomerella cingulata* (Atk) Spaulding and von Schrenk.[1] The imperfect stage on apples was first described by Berkeley as *Gloeosporium fructigenum*, but he had previously described a ripe rot of grapes under the name *Septoria rufo-maculans* which he later changed to *Ascochyta rufo-maculans*. Since it was found later that the fungus causing ripe rot of grapes was identical with the one causing bitter rot, *Gloeosporium rufo-maculans* was generally accepted as the proper name of the imperfect stage. The perfect stage of the apple bitter rot was found by Clinton in 1902, both on the fruit and in pure culture. Based on the work of Stoneman on the perfect stage of similar species of *Gloeosporium*, he renamed the fungus *Gnomoniopsis rufo-maculans*. Unfortunately the name *Gnomoniopsis* had been previously used to describe an entirely different group of fungi, and it became necessary to change the generic name. This was done by Spaulding and von Schrenk in 1903 when they renamed the fungus *Glomerella rufo-maculans*. The fungus described by Atkinson as *Gloeosporium cingulatum* was renamed by Stoneman *Gnomoniopsis cingulata* when she found the perfect stage, and since this fungus was found to be identical with the bitter rot organism and since her description preceded that of Clinton, *Glomerella cingulata* became the accepted name.

Hosts. *Glomerella cingulata* has been reported as a parasite on a large number of plants widely separated botanically. It causes a canker of privet, a ripe rot of grape, pears, peaches, and many other fruits. It has been inoculated into stems, leaves, and fruits of many plants, where it behaves as a more or less virile parasite. For this reason the various host plants of the fungus will not be listed. It causes a definite well-known disease on apples and pears (bitter rot), grapes (ripe rot), and privet (anthracnose).

Morphology. The *mycelium* of *Glomerella cingulata* consists of rather narrow, sparsely septate threads which are at first hyaline, but later, both in the host and in culture, take on an olive tint. Hyphal growth is very rapid under favorable conditions.

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[1] Class Ascomycetes, Order Sphaeriales, Family Gnomoniaceae.

Acervuli are formed both in culture and on the host. These consist of a basal cushion or stroma several to many layers of cells in thickness. This structure in the host tissue is somewhat cup-shaped, owing to the fact that a thicker layer of cells is produced near the outer edge of the cushion. The acervuli develop just beneath the cuticle, which is ruptured by the upward growth of the developing mass of conidiophores and conidia.

Conidia are readily produced in culture on short-branched conidiophores, which may not be in an acervulus but arise from a thickened mass of hyphae. On the fruit the *conidiophores* consist of a columnar mass of elongated cells extending upward from the cushion previously described. There is a tendency for these to grow from the periphery inward so that the whole acervulus, together with the cuticle of the host, gives the appearance of a flattened pycnidium rather than an acervulus.

The conidia in mass are pinkish and are held together in a viscid mass which under dry-weather conditions becomes firm and horny in character. Under the microscope the spores are colorless and nonseptate. They are typically oblong, but vary considerably in shape and size, a feature which is characteristic of *Gloeosporium*. The average may be given as 12 to 16 by 4 to 6 μ, but they may vary in length from 10 to 35 μ and in width from 3.5 to 7 μ.

On germination a septum is often formed across the conidium. The germ tubes, one or two from each conidium, grow very rapidly, and for some time no septa are formed. In contact with a surface or under adverse conditions a knoblike structure is formed at the end of the germ tube. This is called an *appressorium* and is supposed to function as an anchoring organ. Most of the protoplasm of the germ tube migrates into the appressorium and is quickly cut off by a septum. The contents of the appressorium darken soon after the septum is formed.

Species of the genus *Gloeosporium* in contrast with *Colletotrichum* are not supposed to have bristlelike hairs (*setae*) in the acervulus. However, it is not at all uncommon to find a few of these present, especially in culture.

The *perithecium* of *Glomerella cingulata* commonly occurs in culture and is occasionally found on overwintering fruit. It has been found that only certain strains produce the perithecia abundantly in culture. Most cultures produce black stromatic masses, 2 to 8 mm. in diameter, on which perithecia-like bodies are to be found, but these frequently remain sterile. The fertile perithecia from the black stromatic masses described above are usually globose or subglobose, without a distinct neck, but occasionally are beaked, black in color, and measure 125 to 250 μ in diameter. They separate from the stromatic mass with ease when this is crushed.

The *asci* vary greatly in size and shape but average about 75 μ in length and 8 μ in diameter. They range from 50 to 110 μ in length. They are clavate in shape, tapering toward either end and with a short pedicel at the lower end. The walls are thin and easily ruptured. The *ascospores* are often borne in two ranks in the ascus, but this is by no means a constant arrangement, since a uniseriate or irregular arrangement is often seen. The eight ascospores are hyaline, slightly curved, nonseptate, 12 to 22 by 3.5 to 5 μ. The ascospores resemble the conidia and consequently are easily overlooked when the latter are present. Ascospores germinate readily and much in the same manner as the conidia, even to the formation of a septum previous to germination.

Disease Cycle. The fungus causing bitter rot may survive the winter in a number of ways: (1) as stromatic masses in the mummied fruit, giving rise to conidia or ascospores in the spring; (2) in cankers, either produced directly by the bitter rot organism (rare) or in cankers produced by other agents; and (3) in dead bark crevices or other protected places, where it leads a strictly saprophytic existence. Since conidia are produced in abundance from all these sources, it is probable that the perfect stage plays little part in primary infection.

Mummied fruit either on the ground or retained on the tree is a common focus of primary infection. Both conidia and ascospores are produced under such conditions, and it has been observed frequently that where mummied fruit is left on the ground, infections start on fruit of the lower branches. The mummied fruit on the tree is probably responsible for the majority of primary infections.

It is a matter of common observation that bitter rot starts in a definite area and frequently in certain trees in an orchard year after year. This would indicate a perennial source of inoculum, but often careful search fails to reveal definite bitter rot cankers. Such conditions can be explained by the fact that favorable conditions exist in such areas for the continuous saprophytic development of the fungus.

After primary infection the disease often spreads with remarkable rapidity throughout the orchard. Since the spores are produced in viscid masses, their rapid distribution must be explained on the basis of washing and wind-blown mist combined with favorable weather conditions for rapid germination and infection. Insects and birds probably play some part in spore dissemination but are of minor importance. The exuding spore masses sometimes form rather definite tendrils, or threads, on the apple. These may be broken off and be wind-carried during dry weather. The conidia when protected by the gelatinous material of the exudate retain their viability over a long period, although they lose this power when not so protected.

Infection does not occur until fairly late in the growing season and as the fruit approaches maturity. This is due in part to the fact that the green fruit is more resistant than ripe and in part to the fact that the bitter rot organism requires rather high temperatures for rapid development. On early varieties, such as Transparent, maturing in late June, bitter rot may be found by the middle of this month. As a rule the primary infections are rarely observed before July, while the disease usually does not reach epidemic proportions until August or September. Hot, "muggy" weather over a period of a week or 10 days during these months is likely to result in serious bitter rot conditions in the region where the disease is of common occurrence.

Spores alighting on the surface of the apple germinate within a few hours if moisture is present. The appressoria are formed, anchoring the spores to the surface. This fungus is able to infect uninjured fruit by direct penetration, probably through the production of an enzyme which dissolves the cuticle. Under favorable conditions some infections may occur as early as 5 hours after inoculation, and within 10 hours most of the infections have taken place. After penetrating the cuticle the mycelium grows between the cells and, again by enzymatic action, breaks down the tissue, thus causing the characteristic rot.

Reinfections continue throughout the season during wet weather until either the entire crop is ruined or until cold weather checks the development of the conidia. During this period, the fungus also becomes established on broken branches, wounds, old cankers, dead twigs, and fruit spurs.

Relation to Environment. Bitter rot is often spoken of as a hot-weather disease, and epidemics usually occur only during the hot months of the year. The rot develops very slowly at temperatures between 60 and 70°F. Thus, not only is germination delayed at lower temperatures, but also conidial production is decidedly retarded on the rotted spots.

Moisture also plays an important part in the development of this disease. While germination and infection are rapid, the dissemination of the spores is dependent upon rains. Furthermore, at the season of the year when infection occurs, rapid drying of the fruit is likely to result from the high temperatures and the character of the fruit surface. Conidia remain viable over a period of several weeks when retained in the gelatinous matrix of the pustules, but when washed free of this and allowed to dry, they are not viable after 24 hours.

Variability in culture, pathogenicity, and in production of perithecia is marked in *Glomerella cingulata*. It is evident that many strains of this species occur, and this fact may explain the reason for its epidemic behavior in certain areas and its weak pathogenicity in other equally favorable regions for its development. It may also explain why lists of susceptible and resistant varieties of apples and other host plants presented by different investigators do not always agree. The biologic forms of this species have not been thoroughly investigated so far as the apple is concerned, although Edgerton as early as 1908 recorded two forms of the fungus from apple which had different temperature relations, cultural characters, and pathogenic behavior.

Control. Sanitation is of considerable value in the control of bitter rot. This is evident when one considers the sources of primary infection. Mummied fruit, regardless of the original cause of its death, may harbor the bitter rot fungus and consequently should be removed. The same is true of blighted twigs and fruit spurs. In an area of the orchard where bitter rot appears year after year, special attention should be given to the removal of all dead wood and the cutting out of cankers. All rotted fruit should be removed from under the trees.

The prompt removal of the first rotted fruit by careful hand picking has been found to be a valuable supplement to the spray program. Such fruit should be removed before the formation of the spore pustules, collected in a closed utensil, and carried to a distance from the orchard.

Experiments on the control of bitter rot by spraying were started soon after the introduction of Bordeaux mixture in this country. Spraying became a standard recommendation during the first half of the present century. The results were not always favorable, and in dry summers excessive injury to the trees resulted. The fact that epidemics of bitter rot are sporadic in their appearance and that many orchards even in the bitter rot area rarely suffered from the disease led to the virtual abandonment of a set spray schedule as a general recommendation, except in problem orchards. Recently some of the organic fungicides such as captan have gained favor because of their greater efficiency and lack of injury.

Varietal selection decidedly reduces the chances of loss from bitter rot, but on account of the known variability of the causal fungus, too much reliance cannot be placed on this method of control. In a section where bitter rot is known to be prevalent, preference should be given to varieties regarded as resistant by the growers of that section.

Frequently bitter rot appears near harvesttime, and on harvesting it is found that a few diseased apples are brought in to the graders. Under such a condition it is probable that further inoculation has occurred and that rot will develop in transit. Since the rot does not develop at temperatures below 50°F, it is essential that such fruit be placed under refrigeration conditions as soon as possible after harvest.

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6

Black Rot (Frogeye Leaf Spot)

Several common names have been applied to this disease based on the organs attacked. For example, the disease on the fruit is called "black rot," on the leaf "frogeye leaf spot," and on the limbs "New York apple tree canker," or "canker." The rot on the fruit has also been called "ring spot," "brown rot," and "blossom end rot," while the canker is referred to as the "black rot canker."

History. In horticultural literature many references can be found to "rots" of the apple. Unfortunately it is not always possible to identify these rots from the descriptions. For this reason no mention will be made of these early records, although it is safe to assume that black rot occurred many years before it was positively identified by scientific workers. The first report of the rot on the fruit was made by Peck in New York in 1879. In this same year specimens sent by French, from Carbondale, Illinois, were identified by Peck. Frequent references can be found to the rot in various parts of this country and Europe between 1890 and 1900, when mycologists were busy identifying the various fungi causing diseases of economic crops. It is evident from these reports that the disease was generally prevalent throughout the regions of the world where mycologists were active.

The first reference to the leaf spot was made by Alwood, who in 1892 stated that it had been observed by him for several years previous as causing some damage, with an especially severe outbreak in 1891. He ascribed the disease to another fungus which is commonly found on the lesions produced by the black rot fungus. In fact it was not until 1908 that Scott and Rorer positively determined the cause of the leaf spot, although Clinton as early as 1902 stated that the leaf spot in Illinois was due to *Sphaeropsis malorum*. During the period between 1890 and 1900 frequent references were made to the leaf spot from various sections of the country, indicating that it had a wide distribution.

In connection with the leaf spot considerable difference of opinion developed among investigators as to the causative organism. In the early investigations the presence of species of *Phyllosticta*, *Coniothyrium*, *Alternaria*, and *Corynium* led to the belief that these alone or in combination might cause the disease. The results of inoculation experiments by Scott and Rorer in 1908 and by Crabill in 1915 established the fact that *Sphacropsis malorum* was the primary cause of the typical frogeye leaf spot and that the other fungi were secondary invaders.

Paddock, working in New York, was first to call attention to the canker form of black rot and to describe it in detail. In 1899 he established the fact that the organism isolated from the cankers was not only parasitic when inoculated into live bark, but was also identical with the organism causing the black rot of the fruit, and by cross inoculation proved this point. The monographic treatment of Hesler in 1916 reporting the results not only of his own experiments but those of the earlier investigators summarized and rounded out the experimental work up to that date. In 1913, Hesler had found the perfect stage of this pathogen on apple twigs and proved its connection by cultural and inoculation methods. He also reported the perfect stage as occurring on witch hazel and white oak.

Stevens in 1933 and 1936 reported the results of an extensive study of the taxonomy of the black rot fungus and related species. He came to the conclusion that at least two species occurred in the United States and Europe, one forming mainly dark spores in the pycnidium as contrasted with the other with hyaline pycnospores, somewhat smaller in size and more regular in shape. Of the two species the dark-spored one occurs most commonly in the eastern United States and in southern Europe, while the light-spored one is mainly in the northwestern United States and western Europe.

Foster in 1937, in studying a number of collections from different sections of the United States, again emphasized the great variability of *Physalospora obtusa.* He found, for example, that leaf-infecting isolates occur less frequently in the upper Mississippi Valley than in the eastern states.

The control of the leaf spot form of black rot received special attention by Brooks and DeMeritt in New Hampshire in 1912 and by Walton in Pennsylvania in 1920.

Geographic Distribution. The fungus causing this apple rot has been reported from practically all regions of the Temperate Zone. Its wide distribution may be accounted for by the fact that it is common on many plants other than the apple and is able to lead a saprophytic existence for indefinite periods in twigs, old bark, etc.

Apple rot and leaf spot have also been reported over a wide range, not only in this country but in Europe. Leaf spot seems to be most prevalent and destructive in sections of the eastern states, while the canker form is reported as especially prevalent in New York. Neither the leaf spot nor the canker are common west of the Rocky Mountains, and the fruit rot is very rare in the fruit sections of the northwest.

Economic Importance. Losses from black rot result from (1) rotting of the fruit, (2) injury to the tree through defoliation, and (3) loss of limbs by cankers. Of these the greatest loss results from defoliation, since this not only results in fruit of inferior quality in the season of defoliation but also reduces the crop the following season. Loss to fruit is not to be compared to the loss from bitter rot in commercial orchards, since most of the black rot occurs on fruit injured by worms or otherwise wounded and such fruit would be discarded in packing. In some years considerable loss occurs through calyx end infection. In common storage, rot may be rather serious where the fruit has received a certain amount of wounding through insects and handling, but under modern commercial conditions of insect control and rigid sorting of fruit, the rot is of minor importance in storage.

It is difficult to estimate the injury due to cankers since many factors are involved. While the black rot fungus is generally present on dead twigs and bark, it is not an active, destructive parasite under normal conditions. In some localities, however, the actual damage to limbs may be great. Paddock, in New York, cites a case in which 30 acres out of 80 were ruined by this disease and the remaining 50 were seriously injured. Hesler observed an orchard of 350 trees and found 33 per cent with from one to three dead limbs each. The loss from black rot canker is most serious in winter-injured or neglected orchards and is rarely found on vigorous growing trees.

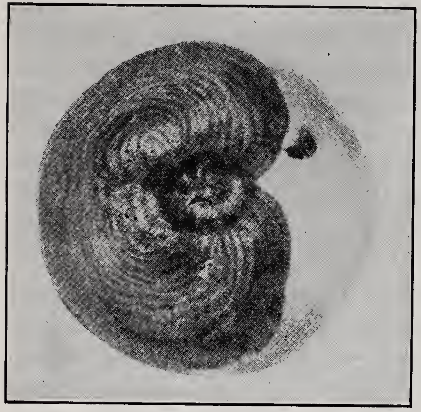

Fig. 21. Black rot on apple fruit showing alternate light and dark bands (these are not always evident).

Symptoms. Black rot on the *fruit* usually appears around a wormhole or some other wound. Frequently it originates at the calyx end where splitting or spray injury opens up the way for infection. Ordinarily only one spot occurs on a fruit, thus differing from bitter rot. When the rot first becomes evident the spots are brown, and this color may not change with an increase in the size of the lesion. As a rule, however, the area where the rot first develops turns black. Frequently a series of alternating concentric rings is formed, the darker bands being a deep mahogany brown to black (Fig. 21). In all cases the rotted apple finally turns black. The rotted tissue is somewhat firm and inclined to be rather leathery when the rot occurs before the apple is completely mature. The taste is not unpleasant as in the case of bitter rot, and the musty odor of blue mold rot is absent. The rotted area remains level with the healthy surface of the fruit for some time, thus differentiating it from the bitter rot lesions which show a saucerlike depression. After the fruit is completely rotted the tissues collapse owing to drying, and a wrinkled black mummy results.

When the black rot spots start on one side of the calyx cup or on the side of a wormhole, the outline is not circular as in the case of bitter rot.

Sooner or later black, pimplelike fruiting bodies appear on the surface of the rotted area. There is a great variation in the stage of rot at which these appear. In some cases they may be seen by the time the spot is ½ in. in diameter, while in other cases they do not appear until the fruit is completely rotted or not at all. Their distribution and number may also show decided variations. The rotted fruit is not inclined to drop from the tree and often remains through the winter months as a black wrinkled mummy. In storage the rotted fruit is inclined to become watery, but the flesh, unless invaded by other organisms, is firm in comparison with the soft rots.

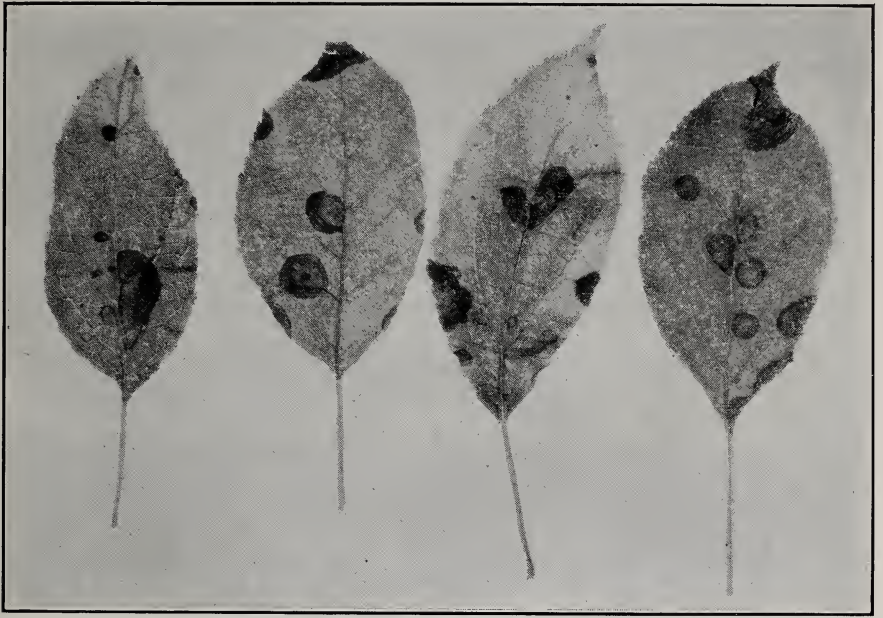

FIG. 22. Black rot (frogeye) on apple leaves.

On the *leaf* the first evidence of the disease is the appearance of few to many small purple specks with a rather indefinite outline. Ordinarily, these are first evident about 1 to 3 weeks after petal fall, although they may appear earlier. The spots enlarge to a diameter of about ⅛ to ¼ in. The margin of the spot remains purple, while the central area turns to a brown or yellowish brown. At this time the outline of the lesion is almost always circular, but later it takes on a lobed appearance due to an irregular secondary growth of the pathogen. The spots at this time show a purplish border with a zone of dark brown about a central area varying from light brown to gray. The entire effect is responsible for the popular name "frogeye" (Fig. 22). The appearance of the leaf spot is often modified by the secondary invasion of various fungi, and especially by the effect of spray materials on the tissues immediately surrounding the spots.

Small black hemispherical dots may be seen near the center of the lesions on the upper surface of the leaf, but these are not always evident.

The effect on the leaf varies with the severity of infection and the variety attacked. In some varieties, such as Jonathan, a leaf showing only a few spots will turn yellow and drop off. Apparently, defoliation is less pronounced when the infection occurs late in the season.

The following description of the symptoms on the *limbs* is quoted from Hesler:

> In the earlier stages of the formation of a canker, the bark is slightly sunken and reddish-brown in color. The diseased area slowly increases in size and darkens, and, although not conspicuous at a distance, the spot is readily distinguished from healthy tissue on closer examination. Some lesions remain very small, measuring only a few centimeters in their longer diameter; in such cases the canker usually dies out at the end of the year. Where the injury is larger, the diseased spot enlarges from year to year for a distance of a meter or even more.
>
> It is often observed that a canker is merely a superficial roughening of the bark. In other cases the bark is killed to the wood and becomes conspicuously cracked.
>
> The discolored area may extend over a considerable surface; or regardless of its size, a crevice may appear at the margin, limiting temporarily at least, the extent of the lesion. Further spread of the pathogene results in the formation of a prominent spot, which soon forms a second line of demarcation between the healthy and the diseased tissue. Repetition of this process from one or more points at the margin occurs, thus producing a lobed appearance; or the spreading may arise from all points about the first marginal crack, so that a series of concentric crevices is developed.

The bark clings firmly to the wood in recently killed areas, but after a year it cracks and finally can be easily removed. Often a callus is formed about the margin of the wound, and gradual healing and covering of the area occurs.

Pimplelike protuberances may be seen on the surface of the cankers, but these are not always evident. When present they are usually very numerous. They are usually more conspicuous on small twigs where the black rot fungus has developed saprophytically. Frequently secondary fungi invade the cankers and thus confuse the symptoms described above. Beneath the rough exterior the bark is dry, hard, and tough as compared to the brittle, soft bark in blister cankers. Rather definite alternate layers of dark and light bark are sometimes evident, but there is no evidence of the mottled appearance seen in blister cankered bark. Black rot cankers are easily confused with those caused by other agencies. Fire blight cankers when invaded by the black rot organism show the definite pimple-like pycnidia and sunken bark characteristics of the black rot cankers. Superficial cankers caused by *Myxosporium* and other agencies may show an abundance of the fruiting bodies intermingled with those of other fungi.

Causal Organism. The nomenclature of the organism causing black rot is very much confused because of the fact that it has been collected and described under different names from a large number of hosts, both in the imperfect and perfect stages. The imperfect stage has been described as one or more species of the genera *Sphaeria, Diplodia, Melogramma,* and *Sphaeropsis.* In 1879, Peck described the fungus on apple as *Sphaeropsis malorum,* which name has been generally used for the imperfect stage since that date. The perfect stage of the fungus was first discovered in America on apple twigs by Hesler in 1913. He determined that it belonged to the genus *Physalospora.* Arnaud in France in 1912 had described a *Physalospora* which seemed to agree with this fungus, and consequently Hesler accepted Arnaud's name *Physalospora cydoniae.* The combination *P. malorum* was proposed when it was found that Arnaud's *P. cydoniae* differed in some respects from the perfect stage of the common black rot fungus in America. In 1933 Stevens, as a result of careful study of type specimens, came to the conclusion that the earliest description of the fungus had been made by Schweinitz in 1832, who named it *Sphaeria obtusa.* In 1892, Cooke in examining specimens from the Schweinitz collection, found the perfect stage of this fungus and transferred it to the genus *Physalospora.* Thus the correct name would become *P. obtusa* (Schw.) Cooke,[1] provided that the two associated stages belonged to the same fungus. This was proved by Stevens from fresh material collected both in England and America, from which cultures were obtained. On the basis of his findings, Stevens also was of the opinion that two species of *Physalospora* were associated with the black rot of apples. The one common in western Europe and northwestern United States he named *P. mutila* (Fries) N.E.S. The synonomy of *P. obtusa* given by Stevens shows 62 synonyms for the imperfect stage and 4 for the perfect stage, while *P. mutila* has at least 15 synonyms. This excessive number of synonyms has arisen partly from the fact that the saprophytic stage occurs on some 75 plants and partly because of the rather wide variation in the morphological characters of the fungus under different conditions. It seems best, therefore, to adopt the name *S. malorum* when referring to the fungus in its relation to the black rot disease, and *P. obtusa* for the saprophytic perfect stage.

Hosts. Stevens lists 75 plants on which *Sphaeropsis malorum* has been found. It would seem, therefore, that the fungus will grow saprophytically and fruit on almost any plant having a woody texture if conditions are favorable. As a parasite, however, it seems to be limited largely to the pome fruits, apple, pear, and quince. The leaf spot is known only from the apple.

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[1] Class Ascomycetes, Order Sphaeriales, Family Pleosporaceae.

Morphology. The mycelium is composed of branched, septate, rather broad hyphae, which at first are hyaline but later turn to an olivacious to dark brown color. In culture the growth assumes an olive-green color, which later turns black.

The *pycnidia* vary somewhat as to shape but in general are globose or subglobose. They are usually borne singly but may be united in a stroma. They are from 200 to 300 μ in diameter. There are two distinct walls, the outer composed of dark, thick-walled cells, more numerous around the top of the pycnidium, the inner of thin-walled hyaline cells from which develops the clavate, or cylindrical, sporophores. The ostiole is always present. It may vary in length according to the position of the pycnidium in the tissue.

Variations in size, color, shape, and septation of the *pycnospores* have been recorded. As a rule they are nonseptate, but one or more septa exceptionally occur. They range in length from 16 to 36 μ and in width from 7 to 10 μ, but average about 12 by 25 μ. In shape they are usually ellipsoidal, but may vary to pyriform or globose. The color of the mature spore is usually brown with a slight greenish tinge. Hyaline, immature spores are common, and this together with the common presence of two-celled spores has led to some confusion at times in the classification of the fungus.

The *perithecia* are not commonly observed. They occur on dead bark of the apple and other plants and where present are abundant. Frequently they are interspersed with the pycnidia. The perithecia are very similar in structure to the pycnidia both as to size, shape, and character of the walls. For this reason a separate description is not deemed necessary. The abundant *asci* are broadly clavate, 21 to 32 by 130 to 180 μ. The walls are rather thick, especially at the apex. The *ascospores* are ellipsoidal, continuous, hyaline to greenish yellow, measuring 11 to 15 by 23 to 34 μ. While typically eight to an ascus, variations of two or four are not uncommon. The ascospores when first formed are spherical and uninucleate. These elongate into spindle-shaped spores, which later become multinucleate and ellipsoidal.

The perfect stage has not been found on the fruit or leaves. It is probably more commonly present on old bark and blighted twigs than was supposed and has been overlooked because of its resemblance to the pycnidial stage.

Disease Cycle. The life history of the black rot organism in relation to its parasitic phase is complicated by the undoubted presence of a number of distinct strains. In general, it has been found that the strains from hosts other than apple will cause rotting of apple fruit. The rate of rotting varies widely but is fairly consistent with the various strains. Thus it appears that regardless of the source of the spores, fruit rotting will develop under favorable conditions. As mentioned earlier, the relation of various strains to leaf spot infection has received some attention, and it has been pointed out that there is no correlation between the ability of strains to induce leaf spotting and rot of fruit. Hesler was less successful in producing cankers on apple from strains obtained from wild hosts than from those isolated from apple, but here again the results are inconclusive. It is safe to assert that the presence of the fungus on wild hosts may influence the incidence and abundance of black rot in an orchard, but the fact that the organism is so generally prevalent on the apple itself would indicate that the removal of other plants would have very little influence on control.

The fungus can overwinter in a number of ways. It may be present in the mycelial form in dead bark, dead twigs, or mummied fruit. The pycnidia containing viable pycnospores are very common both on the mummied fruit and on cankers and dead twigs early in the spring. Also, the fungus may overwinter in the perfect stage in the bark or blighted twigs and may account for considerable initial infection.

Undoubtedly one of the most common sources of inoculum for leaf infection is blighted twigs. It is common to find in early spring cone-shaped areas of the leaf spot beneath fire-blighted twigs on which the pycnidia and perithecia of the black rot organism have developed. The same phenomenon may be seen in the case of mummied fruit hanging on the tree.

When the pycnidia are wet the mature spores are forced out of the ostiole in the form of a coil, which may extend ¼ in. above the surface of the bark. As many as 1,500 spores have been counted in a single coil. When water comes in contact with these coils, the spores separate with a violent motion which may carry individual spores or masses some distance from the surface of the coil.

Spore discharge in the spring is largely conditioned by the maturity of the pycnidia and perithecia, temperature, and moisture. The heaviest discharge of spores occurs around the blossoming period in the spring, but may continue at intervals throughout the summer.

Infection studies by Walton in Pennsylvania showed that over a 2-year period most of the leaf infection occurred between the 9th and the 25th of May. Hesler records about the same dates for spore discharge in New York.

Spore dissemination seems to be due largely to washing and wind-blown mist, although miscellaneous insects may play a part, since the gelatinous nature of the spores would cause them to adhere to the bodies and legs of any insect crawling over the discharging pycnidia. The prevalence of black rot around wormholes may be due in part to the fact that flies and ants often visit the freshly wounded areas on the apple.

Germination of the pycnospores is rapid under favorable temperature and moisture conditions. Recently matured spores will germinate within 5 or 6 hours, although overwintering ones may require longer periods. The most favorable temperature for spore germination is about 75 to 80°F, while at temperature of 60°F they germinate so slowly that infection is not likely to occur. The incubation period is from 2 to 4 days in the case of leaf spot, from 2 to 9 days for bark lesions, and about 48 hours for the fruit rot. *Physalospora obtusa* is primarily a wound parasite and will not infect uninjured fruit or bark. Leaf infection is generally through the stomata on the underside of the leaf. After invasion through the stomata the toxic material produced by the fungus acts in advance of the mycelium and the cells collapse. Stimulation of certain tissues beyond the area of dead cells results in the formation of abnormal tissues, and a certain amount of hyperplasia results. This tissue is in turn invaded, and the process is repeated, thus giving rise to the alternate light and dark concentric rings. The extent of the invasion does not appear to be strictly limited since an individual spot may be ½ in. or so in diameter by the end of the season. The necrotic tissues are soon invaded by secondary fungi such as *Phyllosticta* and *Alternaria.*

Fruit rot usually occurs as the fruit approaches maturity. The spores germinate in the wounded tissue, and the hyphae penetrate the living tissue. The intercellular mycelium advances rather slowly as compared to other rot-producing fungi. The invaded tissue turns brown, and the cell walls are thickened, thus accounting for the firm nature of the rot. The rate of rotting under uniform conditions varies with different strains.

The bark may be invaded throughout or only superficially. In young twigs the wood may also be invaded, but this rarely occurs in older limbs. The general effect is to form a dark, dry sunken necrotic area with a crevice at the periphery. Abundant mycelium is found in the tissue at the edge of the lesion, but after a period of invasion a definite layer of cork cells is formed which cuts off the advancing mycelium. Diseased irregular areas extend beyond this crevice, the mycelium advancing across the barrier where the sclerenchyma fibers cross. A secondary layer of cork cells is formed, and another crevice results. The mycelium appears to be entirely intercellular.

The invasion of blighted twigs by the black rot fungus has not been studied in detail. It is evident that apple twigs recently killed by fire blight are an especially congenial pabulum for the growth and fruiting of the fungus.

Relation to Environment. Black rot is especially serious as a leaf spot where wet weather prevails during and immediately following the bloom. The rather high minimum temperature for the germination of the pycnospores (60°F) sometimes restricts infection. The pycnospores of this fungus are exceptional in having a long period of viability. This accounts in part for the fact that infection can occur at any time during the year when climatic conditions are favorable.

The presence of fire-blighted twigs in an orchard favors the production of black rot pycnidia, and on account of their favorable position extensive primary leaf infections may occur.

Fruit infections result from wounds caused by insects, hail, etc. Injury to the calyx end of the fruit by spraying often results in cracking and invasion by the black rot organism. In some cases it appears that the fungus gains entrance through the open calyx tube of certain varieties, but here growth cracks may account for the prevalence of calyx end infections.

Control. Control of black rot is complicated by the facts that the pycnospores are almost omnipresent and that they are viable over a period of at least a year. The fungus is found on so many wild plants and ornamentals that there is no assurance that removal of dead wood in the orchard will be of practical significance. However, dead twigs and cankers in the tree are responsible in many cases for the heavy primary leaf infection. This is especially true where numerous fire blighted twigs are left from year to year. The orchardist, therefore, should strive to remove dead twigs and limbs and to clean out the dead tissue in any type of superficial canker, thus reducing the quantity of inoculum.

In the control of the fruit rot the most essential thing is to protect the fruit from injury by thorough control of insects and by the use of safe spray materials. Mummied fruit should be removed from the trees. Care should be taken in handling the fruit in picking and packing in order to avoid wounding. The temperature at which the pycnospores will germinate is rather high, and the rot progresses very slowly at temperatures below 50°F. For this reason immediate refrigeration after packing is a good practice. In common storage where temperatures are high at times, special care should be exercised at picking time to exclude all wormy or injured fruit.

Cultivation when practical from the standpoint of orchard management is advised. Vigorous growing trees are not as subject to cankers as are those growing under poor cultural conditions. For this reason orchard practices which maintain vigor are recommended. All pruning wounds should be made in such a way as to promote rapid healing. Weak twigs should be cut back to healthy vigorous wood.

Spraying. Since the time of maximum infection of the leaves occurs during the period of scab infection, special applications for leaf spot control are not necessary except in those cases where the regular spray schedule for scab control is not followed. Bordeaux mixture is not ordinarily recommended in these earlier sprays since fruit russetting may result.

The trend to substitute oil sprays for dormant lime sulfur solutions for the control of scale has raised the question as to whether an apparent increase of leaf spot in some sections might not be due to this change. Powell in 1944 reported a significant decrease in leaf spot where dormant Bordeaux mixture (12-12-100) was applied as contrasted to trees receiving dormant oil alone.

While some varieties, such as Twenty Ounce, are especially susceptible to canker and others, such as Jonathan, to the leaf spot, the use of possibly inferior market varieties because of their higher resistance is not ordinarily recommended. Furthermore, apparent resistance to leaf spotting may not be inherent resistance, but rather due to factors such as the presence or absence of a leaf spotting strain or to fire blight prevalence in the orchard.

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Soft Rot

Most of the rotting in storage, and especially in the markets, is caused by the soft rot organism. This disease is also called "blue mold" on account of the color of the fruiting bodies. It is not confined to apples and pears, but occurs on many other fruits and vegetables.

History and Geographic Distribution. The fungus causing soft rot was described as early as 1809 by Link. Early references to the disease on apples are numerous, but these are often confusing since no distinction is made between the different types of rots. It is safe to say that most of the apple rots occurring in common storage were of the soft rot type. One of the factors which led to the development of cold storage for fruit was the effort to reduce losses from this rot.

Soft rot occurs on apples and pears wherever these fruits are grown or shipped.

Economic Importance. It has been estimated that blue mold causes 80 to 90 per cent of the storage decay of apples. The loss is especially high since it is incurred after the harvesting, packing, shipping, and storage of the fruit. When only good sound fruit is packed and kept under good storage conditions, the loss is not large. Loss from soft rot falls upon the jobber, the retailer, and the consumer, rather than upon the orchardist, although injured fruit is often rotted on the tree by the blue mold fungus.

Symptoms. Soft rot can usually be distinguished in its early stages by the light color and soft, watery texture of the rotted areas. The taste is characteristic, being disagreeably moldy. The odor is perhaps the most reliable method of detecting the rot in packed fruit. A few half-rotted apples can be detected even when in the bottom of a container, and the characteristic strong, moldy odor, and frequently the taste, cling to the apples which have been kept in the neighborhood of the rotted one.

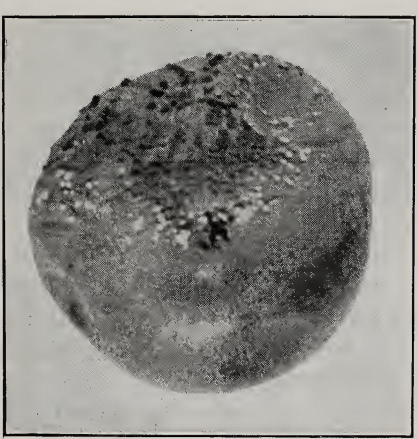

FIG. 23. Soft rot (blue mold) on apple fruit showing green tufts of fruiting bodies.

The rot when started advances rapidly, especially when temperature conditions are favorable. The whole fruit may be rotted in less than 2 weeks after infection. The rotted area does not become sunken; it is usually yellow or light brown in color, very soft in texture, and not inclined to wrinkle. When stored where the humidity is relatively high, the surface of the diseased portion of the fruit becomes studded with the characteristic gray-blue cushions of the fruiting bodies (Fig. 23). Frequently these do not appear on the apple at all, this being especially true where the surrounding air is dry.

Causal Organism. Soft rot is usually caused by the fungus *Penicillium expansum*,[1] but some other species of *Penicillium* may cause a similar rot.

While the perfect stage of *Penicillium* is known, it plays no part in the development of the disease. When placed under moist conditions, the rotted area becomes studded with wartlike fruiting structures called *coremia*. These consist of numerous grouped *conidiophores* arising from the mycelium which permeates the flesh of the fruit. The conidiophore gives rise to one to three main branches, which in turn produce crowded whorls of branchlets on which are formed chains of conidia, the whole structure resembling a small brush. The conidia are green or blue green in mass, hence the name "blue mold." The conidia are elliptical or globose, measuring 3 by 3.3 μ.

The perfect stage is rarely found. The closed ascocarps (cleistothecia) are small, indehiscent, smooth, spherical bodies filled with asci containing four to eight one-celled spores. *Penicillium expansum* and related species grow on all sorts of decaying material and upon numerous fruits.

Disease Cycle. The spores are very resistant to unfavorable weather conditions and consequently may be regarded as always present and able to infect if conditions are favorable. Fortunately this fungus is only weakly parasitic. It is also primarily a disease of ripe fruit, rarely attacking green fruits.

The spores of the soft rot organism fall upon wounded surfaces, immediately germinate, and soon develop branched mycelium in the tissues of the apple, rotting the connecting material of the cells and causing them to become dissociated. This results in the characteristic soft texture of the diseased tissues. These spores are very light and are readily carried by drafts of air in all directions, infecting other fruit. The spores do not become wet when they come in contact with water, but float like dust on the surface. They may be blown into the air from the surface of a drop of water. When fruit is in close contact, the mycelium may pass directly from one fruit to another, provided there is a bruised or wounded area on the second fruit. Sound fruit may remain in contact and partly surrounded by entirely decayed apples and not become infected, although it is rendered worthless by the moldy taste imparted to it. Infection through lenticels is possible, and bruised fruit without evidence of external cracks often becomes infected.

Control. Every effort should be made to avoid injuring the fruit so that it may present an unassailable front to the omnipresent spores. The fungus may gain a foothold through lesions produced by other fungi, through insect injury, or through wounds resulting from the handling of the fruit by pickers, graders, packers, and marketmen. The fruit should be kept as free as possible in the orchard from insect and fungus attack, and in packing, all diseased or injured fruit should be discarded. The fingernail scratches of the picker or packer, or scratches from twigs, rough places on the crates or baskets, projecting nails on the grader, etc., frequently cause wounds sufficient to permit infection.

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[1] Class Ascomycetes, Order Eurotiales, Family Aspergillaceae. Class Fungi Imperfecti, Order Moniales, Family Moniliaceae.

Next to care in picking and packing, the important factor in control is keeping the temperature as low as possible from the time the fruit is harvested until it reaches the ultimate consumer. Actual experiments have shown that *Penicillium* and some other rot-producing fungi are able to develop at temperatures as low as can be obtained without injury to the fruit. But the growth and consequent spread of the rot are very slow at low temperatures and are almost entirely inhibited at 32 to 35°F. *P. expansum*, when allowed to develop at normal temperatures for a few days, will induce rapid rotting after the fruit is transferred to cold storage; thus it is essential to avoid any delay in placing the fruit in cold storage after packing. The great improvements in handling fruit by commercial growers within the past decade have had a marked effect on reducing wastage from this disease.

Addition of certain disinfectants to the wash water used in cleaning apples during grading has proved effective in reducing the amount of fruit rots.

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CHAPTER 3

DISEASES OF POME FRUITS (CONTINUED)

Fire Blight

Because of the early association of fire blight with the pear, a full discussion of the disease will be given under this host rather than under the apple, on which it is probably of more general importance at the present time. It should be remembered in this connection, however, that the commercial pear industry of the entire North American continent, with the exception of certain isolated, well-guarded sections of the extreme West, has been abandoned so far as the growing of quality pears is concerned, because of the ravages of this disease.

Fire blight is the most generally accepted common name for this disease. It is called "blossom blight," "spur blight," "twig blight," and "fruit blight," depending upon the organs attacked. "Blight canker" and "collar blight" are terms used when the disease occurs on the larger limbs or body of the tree.

History. The history of fire blight may be divided into three epochs. The first period extends from the first records of the blight to the discovery of the cause in 1878. The second includes the early investigations on the life history of the organism and the various manifestations of the disease. This period extends to about 1910, when the early investigations of the various phases of the life history as related to the different organs attacked were rounded out. During the next forty years, numerous contributions were made to details of the life history, dissemination of the bacteria, host range, breeding for resistance, and control measures.

Fire blight is undoubtedly of American origin, since it was noticed as early as 1780 by Denning in the Hudson Valley fruit region and was not reported from any foreign country until over a century later. In 1817 Coxe wrote, "Whether the climate of the United States is so well adapted to the cultivation of the pear as the apple, is doubtful in the opinion of some experienced cultivators—that species of blight which is sometimes called fire blight frequently destroys trees in full vigor and health."

Fire blight on both apple and pear seems to have spread to the Middle West with the development of a fruit industry in that section. By the middle of the nineteenth century the disease seems to have been well known by horticulturists in this region. In the 1850 *Proceedings of the American Pomological Society*, Kennicott mentions it as occurring on apple, pear, and quince in Illinois. Fire blight did not reach the fruit-growing sections of the Far West until about 1900, when it appeared in California. Between that date and 1910, it caused enormous losses in the California pear regions and by 1908 had appeared in the Rogue River Valley of Oregon.

No other disease of fruit has given rise to as much discussion as to the cause and cure. The sudden appearance of the disease and its destructive nature were responsible for many theories such as "frozen sap," insects, electric charges, sun scald, etc. It was not until 1878 that Burrill of Illinois definitely established the fact that bacteria were present in large numbers in the diseased tissues of fire blighted twigs, and when the ooze containing "myriads" of these was introduced on the point of a knife into the bark of a healthy tree, blight would result.[1]

In 1883 Burrill named the organism *Micrococcus amylovorus*. Arthur in 1885 confirmed the results obtained by Burrill and extended his experiments, proving beyond question by cultural and inoculation tests that a specific bacterium was the cause of the disease. The problem of the dissemination of the bacteria was undertaken by Waite, who first proved in 1898 by actual experiments that insects play an important part in the transmission of the disease.

Whetzel, in 1906, reported on the results of his studies of the canker form of blight on apples, especially as to their origin and the possibilities of the organism overwintering in these cankers. Jones stated in 1909 that blight had been prevalent in Ontario since 1840 and confirmed Whetzel's results concerning canker development. He was unable to find oozing cankers before blossoming and questioned the observations of Waite that pollinating insects visiting the oozing cankers accounted for the widespread blossom blight.

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[1] As early as 1868 certain experiments and observations by Hull of Alton, Illinois, came close to the results later reported by Burrill. In view of the limited knowledge of bacteria and the limited optical facilities of that time, the observations of Hull were very remarkable. A few significant sentences taken from his reports are here quoted. "In my report to the Illinois Hort. Soc., for 1868 I detailed my experiments in inoculating healthy pear and apple trees with blight. To that report, I will now add that pear, apple, and quince tree blight appear to be identical, since the little cellular growth when taken from one of these trees and introduced into the circulation of either of the others, will induce the disease, to all appearance the same as when it occurs in the usual way." [*Trans. Ill. Hort. Soc.*, 1869, **3**:101 (1870).] In the report for 1870, he states that having received some blighted apple twigs from a correspondent, "We cut from it several small slices of bark, going deep enough to include a thin slice of wood; with these we inoculated several succulent pear shoots, by tying in the pieces of bark as in budding. Soon after inoculating the pear shoots, we were confined to the house by illness and today after a lapse of 34 days were reminded of the experiment on pear shoots. On inspecting them, we found them all blighted. The blight had gone above the inoculated parts, varying from one-half to two inches and descended from three to fourteen inches, further confirming the opinion we have before several times expressed, that the fungi causing pear and apple blight are identical." In another report he speaks of seeing the small "cells" under a "powerful microscope" and refers to these as "fungi."

The third period in the history of blight was characterized by extensive contributions on the origin of primary infection, agents of dissemination, effects on the host tissue, mechanics of infection, migration of the bacteria in the host tissue, and control measures.

Up to this period, it was generally accepted that the bacteria were transferred largely by insects and that the blossom blight was due to the visit of pollinating insects which had previously visited oozing cankers or infected flowers. These assumptions were based on the mistaken opinion that the fire blight bacteria could not infect uninjured tissue except through the nectaries of the blossoms. As early as 1885 Arthur considered the possibility of the bacteria being carried by water, but did not carry out definite experiments. In 1918 Stevens, Ruth, and Spooner published the results of experiments from which they came to the conclusion that bacteria were wind-borne and infection was not always due to insect transmission. They also failed to find insects visiting oozing cankers. Gossard and Walton in 1922 published the results of extensive experiments on fire blight dissemination in which it was definitely proved that abundant primary infection occurs in the absence of insects. They were of the opinion that the inoculum could be carried by water or wind-blown mist as well as by insects. This view was later substantiated by a number of investigators and especially by the experiments and observations of Miller in Wisconsin, Tullis in Michigan, and Rosen in Arkansas (1929).

As early as 1915 Heald called attention to leaf invasion by the blight bacteria and suggested that these entered through the stomata and water pores. If the fire blight bacteria were able to gain entrance through unwounded tissue, inoculation by meteoric water could be explained. The results of various inoculation experiments reported by Miller, Rosen, and Tullis in 1929 proved, beyond a doubt, that young tissues of leaves, blossoms, and twigs could be easily infected in the absence of wounds.

Insect transmission of the disease involved a study of the relation of bees, the chief pollinating insects, to blossom blight. This in turn involved the question of the primary source of the inoculum carried by the bees. Could the fire blight survive in the honey or the hive over long periods? Gossard and Walton, Thomas, and Rosen contributed experimental evidence concerning this point and other phases of insect transmission.

Migration of the bacteria in the host tissue and their effect on the host cells has received special attention by Bachmann, Nixon, Miller, and

Rosen. Nixon in 1928 called special attention to the movement of masses of bacteria in the form of "zoogloeae."

The control of fire blight has received attention since the first disastrous invasion of the disease in the eastern orchards. Many curious remedies were suggested before the discovery of the cause of the disease. Among these may be mentioned the application of lime or wood ashes to the soil, the use of turpentine and coal oil on the blighted parts, dusting with air-slaked lime, root pruning, etc. Based on the relation of the holdover cankers to primary infection, Whetzel, Jones, Stewart, and others recommended careful cutting out and treatment of cankers, blighted branches, etc. In the West where isolated fruit areas favored the eradication of the disease, efforts were made to exclude blight by frequent rigid inspections and eradication.

McCowan in 1929 presented evidence that the application of a weak Bordeaux mixture (2-6-100) would give good control of blossom blight. Rosen reported similar results in 1933.

Many writers have called attention to the problem of resistance and susceptibility in the apple and the pear. The most extensive work on this subject as related to pears was by Reimer in 1925, who has tested thousands of selections of various European and oriental pears.

Geographic Distribution. The disease occurs in all sections of the North American continent where apples or pears are grown, with the exception of a few local areas where conditions are especially unfavorable to its development. Outside of America it has been reported from Japan (1911), New Zealand (1919), and Italy (1924). Its spread in New Zealand was favored by the presence of extensive hawthorn hedges, composed of *Crataegus* sp., which proved susceptible to the disease. The disease has also been reported from Switzerland, Germany, Russia, Transcaucasia, and Turkestan, but these reports have not been confirmed.

Symptoms. The disease may attack any part of the host from the roots to the leaves. In the spring it appears in the form of blossom and twig blight. Infected blossoms suddenly wilt, at first retaining their normal color but soon turning to a dark brown. As the disease progresses down the pedicel, the tissues become water-soaked and dark green in color. Droplets of clear, milky, or amber-colored exudate often appear on the pedicels. The disease progresses into the spur and out into the leaves following the midrib and main veins, which are soon blackened. The leaves wilt, and the entire growth on the spur turns brown (apple) or dark brown to black (pear) (Fig. 26). The leaves cling tenaciously to the stem and often remain attached throughout the year.

Twig blight manifests similar symptoms to those of the blossom blight, except that the progress of the disease down the stem is much more rapid. The recently invaded bark becomes dark green in color with an "oily" appearance, and drops of exudate frequently appear (Fig. 24). The numerous blighted twigs with the clinging dead leaves give the appearance of the tree having been scorched by fire, hence the common name of fire blight. On apples, the leaves remain light to dark brown, while on the pear both the bark and leaves are almost black. Twig blight may also result from girdling some distance below the tip of the shoot following invasion through the spurs or previously blighted twigs or leaves. The gross symptoms are much the same in the two cases.

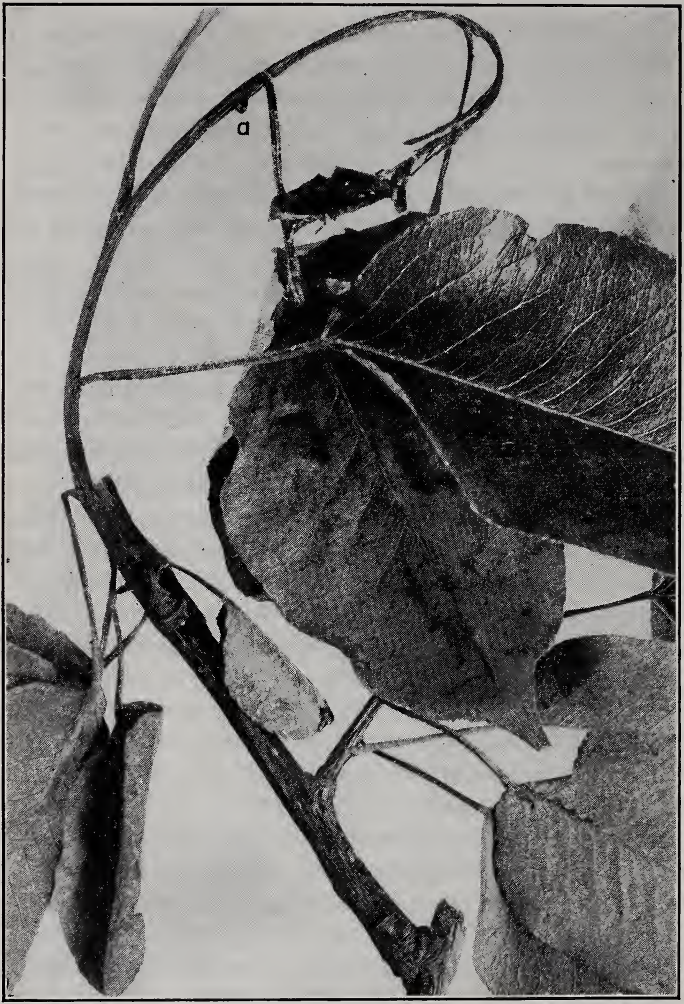

FIG. 24. Fire blight on pear shoot with exuding drop at *a*. Note invasion from midrib on lower leaf.

The downward extension of the blight is finally restricted by various agencies such as maturity of the twigs, and a definite line between diseased and healthy tissue results. The bark above this line becomes dark and shrunken. Often a definite callus tissue appears at this point, separating the diseased from the healthy portion of the twig. Frequently fire blight extends into the larger branches to which the twigs and spurs are attached. These may become entirely girdled with an extension of the diseased area up and down these branches. In other cases a definite canker is produced around the base of the twig. Cankers may also be formed as a result of infection from various other kinds of inoculation. The blight canker, regardless of its origin, has certain characteristics which distinguish it from cankers produced by other parasites. The invaded tissue is at first somewhat water-soaked, with an indefinite outline. Later the bark dries, the area is somewhat depressed, and the surface is smooth (Fig. 25). The dead bark, when cut, is dry and dark brown with no definite flecks or islands of lighter-colored tissue. The dead tissue may extend entirely to the wood or be limited to less than a third of the total thickness of the bark. A definite crevice usually appears around the outer limit of the canker after conditions have stopped or slowed down the tissue invasion by the bacteria.

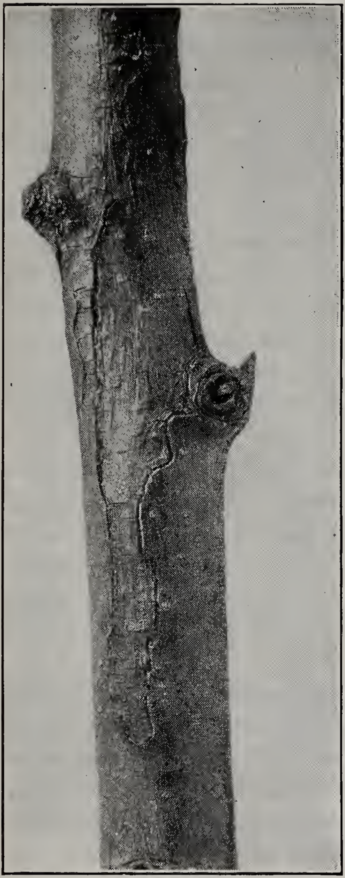

FIG. 25. Fire blight canker showing smooth surface and peripheral crack.

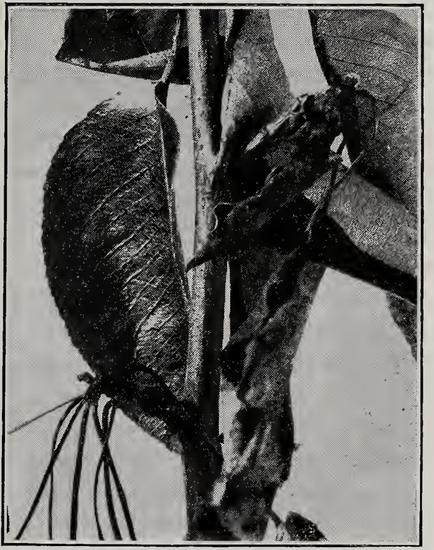

FIG. 26. Spur blight. At lower left, collapse of leaf petiole.

The outline of the canker is usually quite regular, but may be lobed or otherwise irregular, owing to the more rapid invasion of some tissues or to secondary invasion of tissues outside the original canker. A series of crevices sometimes appears as a result of repeated invasions beyond the barrier of the original canker. Invasion of the dead bark of the canker by secondary organisms frequently results in confusion as to the true cause of the original canker. In some cases, the cankered area is not definitely cut off by a crevice, and such cankers are likely to be of the "holdover" type.

Fruit blight frequently occurs on both apple and pear. Usually this occurs before the fruit is half grown. The entire fruit takes on a watery or oily appearance with an abundance of exuding drops of milky or amber-colored ooze. The diseased tissue is superficial and firm, later leathery. In the case of the pear, the fruit turns black, shrivels, and remains attached to the spur, while in the apple it turns to a light brown.

Leaf blight may occasionally occur by direct infection through the stomata, water pores, or wounds. The symptoms are quite different from those where the leaf is invaded through the petiole. Generally the lesions appear on the margin of the leaf and extend inward in a fanlike manner. All the tissue of the leaf may be invaded, and the disease may extend down the petiole into the twig.

Oozing is not a uniform characteristic of blighted tissues. Exudate occurs most frequently during the early spring when the tissues are succulent and the bacteria are advancing rapidly. In some cases no evidence of oozing is to be seen on even recently blighted, tender twigs. The presence or absence of ooze is not a reliable index of the abundance of holdover cankers. Also, cankered areas may "bleed," and yet no bacteria may be evident in the exudate.

Economic Importance. Losses from fire blight cannot be estimated on the basis of annual crop loss. In an orchard where all or most of the blossoms are killed by blossom blight, the loss to the crop may be accurately determined, but when one considers the loss of trees, twigs, or branches, and the influence of this on subsequent crops, the problem becomes much more complicated. Still harder to estimate is the loss resulting from the constant efforts of inexperienced growers to grow fancy pears in regions where blight is certain to kill the trees before they come into bearing.

Some of the losses which occur may be classified as follows: (1) loss or stunting of trees in the nursery, (2) loss of young trees in the orchard, (3) stunting and deforming young trees by frequent blighting of twigs, thus retarding bearing, (4) loss of limbs or entire trees in bearing orchards as a result of girdling by blight, and consequent reduction in crop, (5) partial loss of crop as a result of blossom blight.

In addition to these evident losses another economic factor should be taken into consideration. The pear was at one time a very popular and much used fruit, comparing in commercial importance to the apple. Many people in the eastern United States at present hardly know the taste of a high-quality pear. If high-quality pears could be profitably raised for local markets in the eastern United States, it would add millions of dollars to the income of the orchardists.

Causal Organism. The fire blight bacterium was named *Micrococcus amylovorus* by Burrill. This was changed in 1889 by Trevisan to *Bacillus amylovorus* (Burrill) Trev., which name was generally used, until the adoption of the Society of American Bacteriologists system of nomenclature, when the name became *Erwinia amylovora* (Burrill) Bergey et al.[1]

Hosts. Over 75 species of the Rosaceae are known to be susceptible to the fire blight organism. These comprise mainly species of the genera *Amelanchier* (serviceberry), *Chaenomeles* (Japanese quince), *Cotoneaster* (cotoneaster), *Crataegus* (hawthorn), *Cydonia* (quince), *Eriobotrya* (loquat), *Fragaria* (strawberry), *Heteromeles* (christmasberry), *Mespilus* (medlar), *Photinia* (photinia), *Prunus* (apricot, cherry, plum, prune, almond), *Pyracantha* (fire thorn), *Pyrus* (pear and apple), *Rosa* (rose), *Sorbus* (mountain ash), and *Spiraea* (spiraea). With the exception of the cultivated apple, pear, and quince, none of these hosts are seriously attacked under natural conditions and probably play little part in the dissemination of the bacteria, except in New Zealand where *Crataegus* sp. is of importance.

Host plants other than the members of the rose family (Rosaceae) have not been reported.

Morphology and Cultural Characters. *Erwinia amylovora* is a short rod 0.7 to 1.0 by 0.9 to 1.5 μ. It is motile by peritrichous flagella (Fig. 3, page 6), does not produce spores, forms short chains, and is not capsulate. On agar it forms circular opalescent white, glistening butyrous colonies, with entire margins. It liquefies gelatin very slowly, produces heavy clouding in bouillon or potato broth with a slight granular pellicle; milk is curdled and more or less completely digested; optimum temperature 86°F; thermal death point 110 to 115°F. Acid is produced from sucrose, glucose, lactose, and glycerin.

In the tissues of the host it is intercellular, often producing gelatinous strands, in which form it progresses into the invaded tissue. Exudate in the form of clear amber-colored or milky viscid drops is produced on the host; later these dry to hard flakes or pellets.

Disease Cycle. The origin of *primary infection* in the spring has been the subject of extensive controversy among plant pathologists. The early investigations of Whetzel and others indicated that the bacteria could overwinter in the tissues of the host, especially in a certain percentage of the large body and limb cankers, which were, therefore, called "hold-over cankers." Since these were found to exude in the spring, it was plausible to account for primary infection on the assumption that insects visited this infectious ooze and carried the organism to the blossoms and twigs, thus originating the spring infections. This theory was discredited when it was found that the oozing usually occurred after the appearance of the first infections and that only by accident did insects come in contact with this ooze. Exceptionally, flies were attracted by the exudate, but these did not visit the flowers. It did not seem to be attractive to bees and other pollinating insects.

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[1] Class Schizomycetes, Order Eubacteriales, Family Entobacteriaceae.

The discovery that inoculation could take place in the absence of insects by meteoric water cleared up certain difficulties in accounting for primary infection. But the fact that most of the cankers thought to be of the holdover type occurred on the larger limbs, in such a position that water flowing over them would not come in contact with susceptible tissue, raised another issue. Later investigations showed that the bacteria could survive in a low percentage of the blighted twigs. Various workers reported from 2 to 10 per cent of the twigs of certain varieties yielded the organism in late winter and early spring. The discovery of cone-shaped areas of primary infection below these twigs, following other conditions favorable for a dissemination of the bacteria by meteoric water, pointed to the conclusion that primary infection could be accounted for in the absence of insect vectors. It was further demonstrated that unopened blossoms, young leaves, and new twig growth could be infected in the absence of wounds or insects.

Another controversial point was the question of the possibility of the bacteria overwintering in the beehive and being carried by the bees in the spring to the opening blossoms. It was found that the bacteria will live only a short time (72 hours) in honey. It seems improbable, therefore, that primary infection results from bees carrying bacteria to the blossoms.

Secondary infection may be accounted for in a number of ways. Dissemination by pollinating insects which first visit primarily infected blossoms is regarded as the most common origin of the extensive blossom blight epidemics which occasionally occur, when practically all blossom clusters of certain varieties are blighted. Meteoric water in the form of rains, heavy dews, and wind-blown mist may bring about a wide dissemination of the organism after the exudate has appeared on the primarily infected twigs, blossom pedicels, and leaves. Accidental contact with the exudate or other infectious materials by sucking, chewing, or boring insects accounts for a certain amount of secondary as well as primary infection.

While body, limb, and root blights usually result from the passage of the bacteria into these organs from water sprouts, twigs, spurs, etc., there are some cases where boring insects are responsible. Water may also carry the bacteria into open wounds.

The mode of entry of the bacteria has not definitely been worked out. It was demonstrated early that they may gain entrance through the open blossoms, presumably through the uninjured nectaries, and also through wounds of many kinds. Later, it was determined that infection could take place through the stomata and probably through the water pores in the case of marginal infection. The latter type occurs on leaves with hairy lower surfaces, while the former is more common on the leaves with scant hairs which are easily wet. Older leaves become resistant and are infected through wounds or migration of the bacteria from the infected twigs.

Opened blossoms may be infected through stomata on the inner surface of the receptacle cup (Miller), or through the nectiferous tissue which, according to Rosen, has stomalike openings, or occasionally through the stigma (Pierstorff). Direct invasion of the petals in unopened flowers occurs under very favorable conditions. Twig infection through stomata of the young tissues is similar to that of the leaves.

After invasion the bacteria multiply and migrate into the adjoining tissues. This migration has been carefully studied in recent years and appears to be the result of the formation of definite viscid masses in which the bacteria are embedded. While the bacteria are motile, they do not seem to swim about as individuals, but act in mass. The first few bacteria carried into the susceptible tissues begin the formation of a gelatinous mass within a few hours. From these masses (zoogloeae) as they enlarge, strands (pseudopodia) are pushed through the intercellular spaces of the surrounding tissue, separating the individual cells or groups of cells as they progress. The cell content of the isolated host cells seems to be undisturbed at first, but soon the starch grains disappear and the cells become plasmolyzed and partly collapse. In the leaf the invasion is mostly through the loose mesophyll. In the twig, invasion is first observed in the cortical region of the bark about 10 cells from the surface. From this point it advances in all directions. Invasion of other regions of the twig, such as inner bark, phloem, xylem, and pith may occur, but is slow compared to the rapid spread in the loose cells of the cortex. The last tissues to be invaded are the cambium, xylem, and pith. In the relatively loosely arranged cells of the cortex the progress of the bacterial invasion is very rapid, but is influenced by a number of factors such as the succulency of the twigs and the atmospheric temperature. Most rapid progress takes place at about 80°F and is slowed down at temperatures of 60 to 65°F. The bacteria may travel as much as 2 in. a day through the cortical region. Progress is more rapid up and down than around the stem. The invasion of other tissues lags behind that in the cortex and frequently is confined to this region. This has given rise to the mistaken idea that removal of this outer bark will eliminate the disease.

While the bacteria may be confined to the cortex in recently developed cankers, they soon invade the other tissues and may often be found in the pith of young stems or rarely in the wood of older tissues.

Exudation seems to result from the pressure of the zoogloeal masses. Secondary infections result from the washing of bacteria from the exudate on to susceptible tissues or to the transport of such inoculum by tools, insects, birds, etc.

The bacteria remain abundant in the edge of the advancing cankers, but as soon as the bark tissues die, most of the bacteria die out. While the individual bacteria die after a short period of desiccation, they will remain alive in the dried exudate for months or even years. In nature this long survival does not ordinarily occur, since rains and weathering soon disperse the exudate and expose the bacteria.

Secondary infections may continue throughout the growing season, but during hot, dry weather, with the cessation of terminal growth, fresh infections are few or absent. The progress of the bacteria in the host tissues slows down as the cells become mature and definitely ends in most cases with the formation of a crevice at the edge of the invaded tissue. Beyond this crevice in some cases may be found brown streaks in the live tissue of the bark. Often these extend for an inch or more beyond the border of the canker. In these the bacteria may be found in abundance throughout the following winter. These streaks of invaded tissue are common in cankers of the holdover type and are especially noticeable in Willow Twig apple trees, which variety is especially susceptible to body blight.

Most of the bacteria in blighted shoots and cankers ordinarily die during the summer. With the advent of winter the bacteria which are to give rise to the primary spring infection may be found (1) in a few body or limb cankers, (2) in from 1 to 10 per cent of the larger blighted twigs of certain varieties, (3) in dried exudate protected from excessive weathering, and (4) possibly to a very limited extent in beehives.

Holdover cankers may be more numerous on the pear than on the apple. In New Zealand it has been found that perennial cankers are common on *Crataegus*.

Control. Surgical treatment for the sole purpose of completely eliminating fire blight in an orchard is not considered practical in a region where apples, pears, or quinces are generally grown and where the disease has become established. This comprises most of the fruit-growing area of North America. The exception to this general statement is in the case of areas where apples or pears are grown exclusively by commercial orchardists in an isolated region, such as is found in certain valleys of the Far West. Under such conditions, aided by close cooperation among all the growers and by strict legislative measures and state and county cooperation, it is sometimes possible to control blight by exclusion and eradication. In the regions where farm and city home orchards are distributed throughout the country it is not possible to eradicate the sources of the primary inoculum.

Young trees in the nursery or in the orchard are frequently badly deformed and stunted by twig blight. In a limited way, it is sometimes possible to save these by cutting out the blighted twigs as soon as they appear in the spring. The operator in this case should be supplied with a pruning knife, a bottle of disinfectant (1:1000 mercuric chloride with sufficient glycerin to prevent rapid evaporation), a sponge, and a cloth bag, the mouth of which is kept open by means of a small hoop. As soon as the first signs of wilt appear in the twigs the cutting out should begin and almost daily inspections should be made to discover new cases of blight. The sponge is soaked with the disinfectant, and after each cut the knife is wiped with the sponge. The blighted twigs are placed in the bag to be removed and burned. The twigs should be cut back to the limb from which they arise, unless this will result in a badly deformed tree. Under any condition they should be cut at least 6 in. below the last evidence of wilting or of the water-soaked tissues of the twig. The early removal of the shoots to the main branch often results in another healthy shoot replacing the blighted one so that no great harm is done to the tree. It is rarely practical to remove blighted twigs in this way from trees more than 4 or 5 years of age.

In regions where more of an attempt is made to completely eliminate blight from the orchards, as is the case in sections of California and the northwestern fruit section, a procedure is followed which has as its purpose the complete removal of all sources of inoculum in the orchard. Success in the use of these methods depends upon the care and skill exercised by the operator, and men are trained by the orchardists for this particular work.

Canker Treatment. SCARIFICATION. The object of this method is to remove dead bark and most of the material which will be likely to carry the living bacteria and at the same time to save the cambium for renewal of growth and the quick healing of the wounds. Disinfectants are applied which will not only disinfect the surface but will penetrate into the tissues to such an extent that any bacterial masses not removed will be reached and the organisms killed.

The following directions are given by Day of the California Experiment Station.

> In older parts of the canker the dead or diseased outer bark is shaved inwardly until wide streaks of healthy tissue appear and nearly all of the discolored streaks are removed. At the active margins of the canker, the disease is usually not so deep, but nevertheless the discolored bark should be practically all shaved off. Thick bark such as occurs in crotches and the collar tis-

sue on the opposite side of the branch from the crotch, old bud scars, etc., should be pared away to the fibrous inner layer next to the cambium. Old dry cankers in which the cambium is entirely killed over a fairly large area may be treated in a similar way, provided the cambium is not injured all the way around the branch.

All the wounded surface is then thoroughly painted with a disinfectant, consisting of:

1 qt distilled or rain water
3 qt commercial glycerin
¼ oz cyanide of mercury (16 tablets)
¼ oz bichloride of mercury (16 tablets)

The object of the glycerin in this mixture is to prevent rapid drying and thus allow greater penetration. For treatment of wounds where cankers are cut out such as is recommended for the eastern areas and for disinfecting tools, this formula may be prepared somewhat more cheaply by using 2 qt each of water and glycerin.

After the disinfectant has dried, the exposed wood (where no live cambium remains) should be painted with a Bordeaux oil paint made by stirring raw linseed oil in any of the commercial Bordeaux powders. Another satisfactory method of covering these wounds is the use of shellac and white lead paint. After drying, the surface of the wood is painted with shellac. This is allowed to dry for several hours and then covered with a coating of thick white lead paint.

ZINC CHLORIDE TREATMENT. The object of this method is to kill out the bacteria without the labor of scarification and to save as much of the live tissue as possible. A limited experience with this treatment in the East has emphasized the fact that under different climatic conditions it may be necessary to modify the procedure from that recommended in California. Since the success of the method depends upon a number of factors and careful manipulation, the details of which cannot be given in this brief treatise, the grower who wishes to use this method should consult Circular 20 of the California Agricultural Extension Service, "Pear Blight Control in California." Two solutions of zinc chloride are recommended, one called "43 per cent" and the other "53 per cent," with the following composition.

Formula 1: 43 per cent zinc chloride. Dissolve 6 *pounds* of zinc chloride in the following solvent:

1 gal denatured alcohol
1 pt water
3 oz concentrated hydrochloric acid

Use an enameled kettle. The solution becomes very hot, and any lumps of zinc chloride should be crushed and the mixture stirred with a wooden paddle while hot.

Formula 2: 53 per cent zinc chloride is made with the same solvent to which is added 9 lb of zinc chloride in place of 6. (The 43 per cent solution can be made from the 53 per cent by diluting 1 gal of the 53 per cent solution with 3 pints of the solvent.)

A few directions and precautions may be briefly outlined. The zinc chloride solution should be applied to the surface of the bark and never to scarified or cut surfaces since the solution penetrates rapidly and kills for some distance. The material may be applied at any time of year, but is most effective during the late winter, spring, and summer. The 43 per cent solution should be used on young trees, small branches, small roots and on the lower parts of long narrow cankers on large branches, trunks, or roots. The 53 per cent solution should be used on all large limbs, trunks, roots, etc., except as outlined above. The orchardist can test the probable effect of the material by painting healthy limbs of several sizes in his orchard with the 53 per cent solution. After a week or 10 days the size of the branches injured by the stronger solution can be determined by cutting along the surface of the bark. The tissue about the lenticels will be injured in all cases, but if the inner bark is not killed, it will be reasonably safe to use the stronger solution on branches of corresponding size. Twigs arising from the area to be treated should be cut an inch or more from the branch, and the cut ends should *not* be painted with the zinc chloride solution.

It should be understood that the zinc chloride treatment of cankers is to be used in connection with other programs of sanitation such as the removal of freshly blighted twigs, disinfecting of tools and wounds with the mercuric-chloride-cyanide mixture, etc.

Orchard Management. As a rule blight is much worse on tissues which are succulent. For this reason the grower should attempt to so manage the orchard as to prevent extensive rapid growth of young shoots in varieties of pear and apple especially susceptible to blight. The use of excessive nitrogenous fertilizers, the cultivation of the orchard to promote rapid growth, excessive pruning, etc., should be avoided. Pears and apples should not be planted in the same orchard or adjoining fields. Insects should be controlled by the regular spray practices, and dead limbs and weakened trees as well as cankers should be kept cleaned out to avoid bark beetle infestations. Where blight cankers are common, pruning tools should be kept disinfected.

Varietal Relationships. In any attempt to select varieties of apples or pears resistant to blight, consideration should be given to certain facts which have become fairly well established. Blossom blight, for example, may appear in a severe form in one year on one variety, while the next year another variety may suffer. In this case the time of blossoming, rather than the resistance factor, was involved. Infection is closely related to the growth condition of the trees. Tissues of one variety may be less susceptible than another at the time weather conditions are favorable for infection and thus may appear to be resistant to the disease. All known varieties of cultivated apple, pear, and quince are susceptible to blight in that inoculations into succulent tissue with a virulent culture will produce blight. Resistance to the rapid advance of the disease into the tissues has been demonstrated for certain varieties.

Under natural conditions certain varieties are known to be most frequently blighted. Yellow Transparent and Jonathan apples, among others, show a marked susceptibility to twig blight. Willow Twig in the Mississippi Valley region where it is most extensively grown is especially subject to the body canker type of blight.

Among pears it may be safely stated that no variety of high quality is resistant to blight. Among pears of relatively low quality the Douglas, Kieffer, Old Home, German Sugar, and Estella have been mentioned as resistant, although the Kieffer, which is extensively grown, blights very severely under favorable conditions.

While a great deal of work has been done on the development of resistant pears by breeding and selection, no variety has been developed among the thousands tried at various experiment stations which shows high enough quality and resistance to recommend it.

Species of oriental pears have been tried in the hope that resistance to blight might make them useful for rootstocks and for breeding. Certain selections of the species *Pyrus callaryana* and *P. ussuriensis* have been found to be highly resistant. Old Home, a communis type, which originated in Illinois, is considered useful for growing trunk and framework branches in which more desirable varieties can be topworked.

Spraying. Some success in the control of blossom blight has been secured by the use of one or two sprays of a weak Bordeaux mixture during the blossoming period. Sulfur sprays have no effect on the bacteria. The use of weak Bordeaux does not seem to decrease fruit set and is not especially harmful to bees, probably acting as a mild repellent. Some apples such as Jonathan which are very susceptible to fire blight are severely russeted by copper sprays applied during the blooming period. Antibiotics applied during the blossom period give promise of control. Recent experiments indicate that streptomycin may be of practical value in commercial orchards.

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Crown Gall and Hairy Root

Tumorlike enlargements at or near the ground line have long been known under the general term "crown gall." When these are accompanied by excessive production of roots, the term "hairy root" has been used. To these two types and to various excrescences on other organs such as raspberry and grape canes, other common names, some limited, others inclusive, have been applied: "crown knot," "root knot," "cane galls," "root tumors," "woolly knot," "woolly root," "burr knots," "aerial galls," "plant cancer," etc.

Since the diseases ordinarily called crown gall and hairy root, caused by bacteria and infectious in nature, are found on all the fruit crops with the possible exception of strawberry, no attempt will be made to limit the following treatment to the apple. This disease on brambles manifests certain peculiarities which make it necessary to treat it further in that section. While most of the investigations of the disease in this country have been in relation to the disease on the apple, crown gall is undoubtedly much more virulent and destructive to both peaches and brambles. The extensive investigations of the disease from the standpoint of the apple are justification for placing the main treatment under this crop.

History. Recognition of noninfectious types of galls as distinct from those caused by definite organisms has given rise to some doubt in regard to the accuracy of some of the earlier accounts of the disease. This is especially true in regard to the history of crown gall in America and is further complicated by the fact that some of the earlier observations may have been based on such malformations as those induced by insects and nematodes.

Crown gall has a rather limited history in the United States, since the nature of the disease was such as not to attract special attention. References to what was probably crown gall on roses and brambles can be found in horticultural writings between 1870 to 1890, but no definite scientific investigation of the disease was made until 1890. Attention was first called to the seriousness of the disease in California in 1892 by Wickson and Woodworth. At that time it was recognized as a serious disease of nursery stock. Between 1890 and 1900 a number of publications were issued calling attention to the seriousness of crown gall in nurseries and the danger of transmitting it to the orchards. State nursery-inspection forces had become strengthened during this period owing to the introduction of San José scale and its distribution on nursery stock. Most nurserymen were interested in cooperating with the state and Federal workers in attempting to eradicate any disease or insect which might impair the vitality of the trees and bush fruit when planted in the orchards. Thus arose the extensive practice among nursery inspectors of requiring the destruction of all trees which showed enlargements on the stems or roots of the trees or any hairy-root condition. Since there was no definite method of differentiating between infectious and noninfectious types of galls and since inspectors were seldom thoroughly trained in differentiation based on superficial examinations, thousands of healthy trees were destroyed annually. Partly as a result of the uncertainty of correct diagnosis and partly as a reaction on the part of the nurserymen to the extensive waste involved in the wholesale destruction of trees, there arose a controversy between nurserymen, inspectors, and plant pathologists as to the seriousness of the disease from the standpoint of the orchardists.

Toumey in 1900 demonstrated that the disease was infectious, but regarded the causative organism as one of the slime molds. In 1907 Smith and Townsend announced the discovery of a bacterium from tumors on

Paris daisy, which they demonstrated would cause typical crown gall. This organism was named *Bacterium tumefaciens* and has since been regarded as the cause of certain types of crown gall.

Between 1903 and 1910 Hedgecock investigated the disease in the field using the extensive nurseries in the Mississippi Valley for his experiments and observations. He attempted to classify the galls according to whether they were "soft" or "hard" and divided the hairy-root types into "simple," "woolly knot," "broom root," and "aerial." He studied the environmental conditions under which the galls occurred, but came to no definite conclusion as to their cause, except that his results indicated that infections must occur through wounds and under favorable soil-moisture relations.

In 1910 Smith, Brown, and Townsend published an extensive bulletin on crown gall in which they reported the results of a large number of cross-inoculation tests on a great variety of plants. Isolations were made from several types of galls, including hairy root. Many of the inoculations were negative, but positive results were obtained consistently enough to warrant the conclusion that the organism concerned was responsible for the galls. Morphological and cultural studies were also made of the isolates. The authors called attention to certain variations in cross inoculations and cultural characters of the various isolates, which led them to question whether they were dealing with variations in a species or with different species.

In 1921 Melhus and Maney published the results of a series of experiments on the control of crown gall on apple grafts in the nursery. They found that infection could be easily accomplished by dipping grafts just before planting in a culture of *Agrobacterium tumefaciens*, that cloth wrappers decreased the amount of gall, and that disinfectants materially decreased the amount of gall but sometimes reduced the stand. An 8-8-100 Bordeaux mixture gave no injurious effect and reduced the percentage of crown gall about 50 per cent.

A series of investigations on crown gall was started as a result of a conference of nurserymen, entomologists, plant pathologists, horticulturists, and nursery inspectors in 1923. Between 1925 and 1936 appeared a number of detailed reports by Riker, Muncie, Keitt, Banfield, Siegler, and others in which an attempt was made to differentiate between infectious and noninfectious crown gall and between the various types of hypertrophies classed under the general terms crown gall and hairy root. In 1926 Riker and Keitt published a comprehensive account of their investigations on the differentiation of infectious crown gall and wound overgrowths which were commonly mistaken for crown gall. Their studies also added to our knowledge of the conditions under which both crown gall and overgrowths are likely to develop. The fact that about 90 per cent of the malformations in some nursery stocks were due to wound overgrowths, according to their data, and that these overgrowths were due to imperfect unions helped explain the variable results of experiments in the past and to emphasize the unsatisfactory status of nursery inspection for this disease.

It was pointed out by Hedgecock and others early in the investigation of crown gall that the condition known as hairy root had some rather definite relation to the malformations ordinarily known as crown gall in the restricted sense of this term. Confusion arose in the diagnosis of this type owing to the fact that all variations existed from the abundant formation of fibrous roots as a result of unfavorable soil conditions to definite enlargements of the crown gall type with a few roots arising from them. Smith and others proved that hairy root could be produced by inoculation with what was regarded as the crown gall organism. The question arose as to why a typical smooth-type crown gall would be produced at one time and a hairy root at another under much the same conditions and on the same host. This again gave rise to the suspicion that more than one organism was involved or that there were various strains of the crown gall organism. Smith had called attention to variations in isolates and had expressed the opinion that variations of the pathogenicity of the isolates as well as the reactions of the host might be explained on this basis. Riker and others reported in 1930 that they had found some variation in a number of isolates and, on the basis of host responses and certain bacteriological differences, that the hairy-root organism should be given specific rank, and consequently it was named *Phytomonas rhizogenes*. A further study reported in 1930 on single-cell isolations called attention to the possibility of previous workers having used mixed cultures of the crown gall and hairy-root organism where variable results were obtained. These workers (Wright, Hendrickson, and Riker) were able to show that cultures from single-cell isolates from the hairy-root organisms and from the crown gall pathogene were remarkably constant in their behavior. "Four single-cell isolations were made from a culture that showed a mixture of the characters of the crown-gall and hairy-root organisms and was earlier thought to be possibly a pure culture of an organism intermediate between these. In the bacteriological studies and inoculation tests one of these single cell strains consistently showed the typical characters of the crown-gall organism and the other three consistently showed those of the hairy-root organism." Siegler and Piper in an investigation of the pathogenicity of the woolly knot (hairy-root) type of crown gall confirmed the results of the above workers in 1931, although they held in this and earlier reports that callus overgrowths were not common even in poorly matched grafts when proper precautions were taken to exclude the organisms responsible for infectious malformations.

Patel, by using a special bile agar, was able to isolate the crown gall organism from the soil and reported in 1928 the results of a study of the distribution and longevity of the organism in various soil types. Virulent strains were isolated after 420 days from infested soil in the laboratory, and after 90 days from infested soil subjected to winter conditions out of doors.

In addition to the usual underground or ground-surface types of galls, commonly called hairy root, woolly knot, and crown gall, certain malformations known as "burr knots" are sometimes found on the limbs of apple trees, often in such abundance as seriously to injure the tree. These have been investigated from time to time, since their structure resembled the underground types in some respects. Evidence is still lacking that naturally occurring burr knots are of parasitic origin.

In 1934 a series of publications by Riker, Hildebrand, Keitt, and Banfield reported the results of extensive field and laboratory experiments on the pathogenesis and seasonal development of crown gall and hairy root, together with the relation of wound overgrowths to these diseases. These experiments definitely established the distinction between infectious and noninfectious crown gall and between hairy root (woolly knot) and smooth crown gall by extensive field tests. The relation of various factors such as temperature, moisture, seasonal development of host, and time of inoculation to the incidence of the disease was studied. Another series of papers which reached much the same conclusions were published by Muncie, Melhus, Patel, Sass, and Suit.

Experiments on the "cure" of crown gall by applications of antibiotics have been reported by Brown and Boyle, Ark, Hampton, and others since 1944.

During the past twenty years extensive investigations have been directed toward basic studies on the crown gall organism in relation to cell proliferation and migration. In addition to the importance of these studies to the fruit-disease problem they are interesting in that the organism concerned may prove useful in solving some of the basic problems related to cancer in animals.

Geographic Distribution. It is probable that crown gall has a world-wide distribution, since it has been reported from such widely separated regions as South Africa, New Zealand, Australia, Asia, and South America, as well as the United States, Canada, and European countries. Because of the difficulty of accurate diagnosis, some doubt exists as to the geographic distribution of the infectious types. The disease in the United States seems to be favored in regions of higher temperatures and moisture in apple nurseries, but on brambles crown gall has been very destructive in more northern regions such as Michigan, Minnesota, Wisconsin, and the Pacific northwest.

Economic Importance. Losses from crown gall and similar malformations have been enormous, especially in nurseries where many young trees have to be destroyed after the nurserymen have gone to the expense of grafting and growing the stock. Melhus and Maney estimated that over 2,000 trees per acre were rendered unsalable as a result of crown gall and hairy root. Data compiled by various investigators and reports from nurserymen indicate that an average loss of 20 per cent is a fair estimate. From 15,000 to 20,000 trees are set per acre from which a stand of from 10,000 to 15,000 trees may be secured at the time of digging. If 20 per cent of these are discarded, the loss per acre would be from 2,000 to 3,000 trees. At a low average price of 25 cents per tree, this would entail a loss of from $500 to $750 per acre.

The loss to the orchardist in the case of apples is not very great, because in many cases the trees show little effect on the bearing qualities or growth of the tree. There is some evidence that galled trees are more subject to winter injury. In the case of peaches the loss may be much greater since peach trees with crown gall are either seriously stunted or killed by the first severe winter.

Probably the most extensive loss occurs on the brambles. In some sections of the country it is impossible to grow raspberries successfully on ground in which the crown gall organism has become established. Raspberries with crown gall are especially susceptible to winterkilling.

Symptoms. In some cases malformations due to overgrowths at the graft union are easily distinguished from the "tumor" type of crown gall, but in other cases the symptoms are much the same. It is reasonably well established that trees showing typical hairy root, or woolly knot, in most cases at least, will yield the hairy-root bacteria. However, excessive growth of fibrous roots due to soil conditions is sometimes mistaken for true hairy root. On peach and brambles little difficulty is experienced in recognizing the swellings which are caused by the crown gall bacteria.

Enlargements of roots because of the attack of woolly aphis and occasionally nematodes are sometimes mistaken for crown gall. In the early stage of development the production of a callus growth about fresh wounds may resemble true crown gall, but later the smoothing over of this growth may differentiate it from crown gall of the infectious type.

Galls usually vary in size from ¼ in. to 3 or 4 in. in diameter, but in extreme cases they may be a foot or more across. While typically roughly globular in shape, they may be elongated or irregular in outline. The surface is decidedly convoluted, especially in the young, "soft" galls. In these galls the interior shows a gnarly growth of woody tissue. The very young tumors are composed of light-colored "frosty" tissue resembling fresh callus growth. As they develop, the outer tissue becomes darker and often turns almost black, owing to the decay of the outer cells. Often the tumors become spongy throughout and may crumble or become detached from the root. In galls which do not go through this process of decay from a permanent union with the root or stem, the surface convolutions are less pronounced, and the inner tissues take on a woody character, thus forming the so-called "hard" galls (Fig. 27).

Hairy root, or *woolly knot,* is more often encountered in the nursery than is the smooth type of gall described above. In its early stages this type is similar in character to that of the smooth type in that the first evidence is a convoluted tumor formation composed of light-colored, frosty masses of tissue. From some of the protuberances root primordia appear, and from these fleshy roots are pushed out. These roots are fairly thick and often extend some distance from the basal gall before the secondary fibrous roots are produced. The excessive growth of these fibrous roots gives the woolly appearance characteristic of the disease. While certain types of soil may induce the production of excessive fibrous

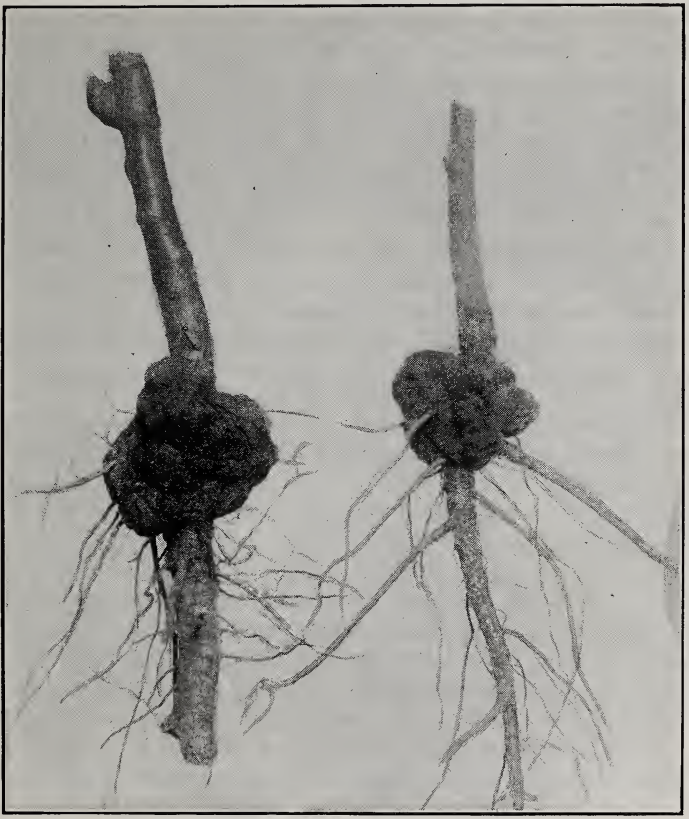

FIG. 27. Crown gall on peach near crown.

roots on healthy plants, these are formed rather uniformly over the entire root system. In the year after infection the basal tumor enlarges, and there is a renewed production of both fleshy and fibrous roots from this and also from the fleshy roots which have survived the winter.

Wound overgrowths, nonparasitic in origin, begin development much as do the infectious type of galls. The frosty callus appears over the surface of the wound and shows the typical convoluted surface. It is more likely to develop on the tip of a cion which extends beyond the root piece (understock) or along the edge of the cion cut where a poor fit is made in grafting. These swellings may progress to the point where they resemble the large smooth-type tumors produced by the true crown gall organism. The enlargements are inclined to be more definite in outline and later to become smoothed over. The interior tissue is decidedly woody and hard except at the surface. A similar type of gall may be produced with wire girdles. This wound overgrowth usually develops a definite bark, while the true crown gall does not form bark tissues. The interior tissue of the true crown galls is composed of a mass of comparatively soft tissue with varying amounts of vascular elements.

Combinations of hairy root, crown gall, and wound overgrowth are not uncommon, thus increasing the difficulty of accurate diagnosis.

Causal Organism. In 1889 Trevisan, on the basis of the results obtained by Corvo and Cubani, named the bacterium associated with crown gall of grape *Bacillus ampelopsorae.* Cavara, in 1897, isolated what appears to have been the same organism, grew it in pure culture, and made inoculations which reproduced the disease. He accepted the name given by Trevisan. Smith and Townsend isolated a bacterium from Paris daisy (*Chrysanthemum frutescens*) and proved the pathogenicity of the organism. This was named *Bacterium tumefaciens* in 1907. Depending on the systems of nomenclature used, this has been called *Pseudomonas, Bacillus,* and *Phytomonas tumefaciens.* In 1942 Conn changed the generic name to *Agrobacterium,* so the accepted name is now *Agrobacterium tumefaciens* (E. F. Sm. and Town.) Conn.[1] Smith and others have recognized that Cavara was probably working with the true crown gall organism but did not accept the name given it by Trevisan because of insufficient proof.

The fact that the bacteria isolated from various hosts and types of tumors varied in their pathogenicity and certain cultural characters was recognized by Smith and later investigators. In 1930 Riker and others, after a differential analysis of a number of strains of *Agr. tumefaciens,* came to the conclusion that the strain commonly isolated from hairy root, or woolly knot, was sufficiently distinct to be given specific rank, and consequently they named it *Phytomonas rhizogenes* [*Agrobacterium rhizogenes* (Riker et al.) Conn]. The bacterium causing raspberry cane gall (see page 331) is regarded as a distinct species (*Agr. rubi*).

Hosts. Elliott lists 63 genera on which *Agr. tumefaciens* is known to produce galls. These genera cover a wide range botanically and include many herbaceous as well as woody plants. So far as fruit crops are concerned, it is most serious on apple, peach, raspberry, blackberry, and grape.

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[1] Class Schizomycetes, Order Eubacteriales, Family Rhizobiaceae.

The host range of *Agr. rhizogenes* has not been accurately determined, since this species was not always differentiated from *Agr. tumefaciens* in the early studies. According to Hildebrand the hairy-root organism has not been reported as occurring under natural conditions on any host other than the apple. This organism was used as a "strain" of *Agr. tumefaciens* by other workers, who successfully inoculated sugar beet, quince, rose, honeysuckle, Paris daisy, balsam, bryophyllum, red raspberry, bean, and sedum. Siegler states that the apple strain *Agr. rhizogenes* will not cause distinct tumors on tomato, while the peach strain of *Agr. tumefaciens* will produce positive reactions on this host.

Morphology and Cultural Characters. The morphological and cultural characters of both *Agr. tumefaciens* and *Agr. rhizogenes* here given are largely taken from the studies of Wright, who made a detailed comparison of these two organisms with *Bacillus radiobacter,* the legume root nodule organism. It is evident from these and other studies that the two species under consideration are closely related to the legume root nodule group.

Agr. tumefaciens and *Agr. rhizogenes* are both small rods approximately 1.5 by 0.5 μ, capsulate, non-spore-forming. *Agr. tumefaciens* is considered by Wright as nonmotile, while *Agr. rhizogenes* is motile by one polar flagellum.

Both organisms form smooth, shining convex colonies with smooth entire edge, opaque to translucent, almost white in color; gelatin not liquefied; Gram-negative; not acid-fast; no nitrate reduction. On litmus milk *Agr. rhizogenes* reduces litmus and gives an acid reaction, while with *Agr. tumefaciens* the milk remains neutral. In the case of sugars, arabinose, xylose, glucose, galactose, fructose, mannose were strongly fermented with *slight* acid formation when *Agr. tumefaciens* was used, while in the case of *Agr. rhizogenes strong* acid reactions resulted from growth in these sugars. Lactose gave no acid for *Agr. tumefaciens* and strong acid for *Agr. rhizogenes,* with weak to medium fermentation. Wright emphasized a calcium glycerophosphate medium with mannitol for differentiating these two species. *Agr. tumefaciens* gave abundant growth in this medium, while the hairy-root organism gave slight to no growth. Serological tests by Wright showed that antisera prepared from each of the two species gave positive agglutination for the homologous species only.

Disease Cycle. Many details of the life history of *Agr. rhizogenes* have been presented by Hildebrand and by Banfield. Since the available information indicates that the two organisms have a similar life history, the following account will be drawn largely from investigations on the hairy-root organism. Of the two organisms concerned *Agr. rhizogenes* appears to be much more pathogenic to the apple. Additional information on the life history of *Agr. tumefaciens* is presented under the discussion of Crown Gall on brambles.

That the bacteria enter the plant only through wounds has been demonstrated by a number of investigators. Bacteria carried into the tissue by needle punctures or placed on the surface of fresh wounds will result in infection regardless of the type of wound. Shallow wounds are not favorable courts of infection. Normal callus growth has been shown to be a barrier to invasion, and ordinarily infection does not occur later than 2 to 4 days after the wound is made, provided insects are excluded. Grafts are most susceptible at the time they are made and, with the formation of callus, become increasingly resistant. That wounds produced by insects feeding upon the roots may increase the amount of crown gall has been demonstrated by studies of Hildebrand and Banfield.

Infection may occur at any time during the growing season. Therefore the amount of natural infection depends upon conditions, such as the presence of wounds favorable for inoculation. The age of apple trees does not influence susceptibility. In spite of the fact that striking varietal differences have been noted in the amount of hairy root occurring in nursery trees, inoculations in 38 varieties of apples by Hildebrand showed that none are highly resistant to the hairy-root organism.

The bacteria after being introduced into a wound multiply first in the watery intercellular medium produced by the rupture of the cells. It is generally agreed that the bacteria are intercellular. Three types of migration of the bacteria into the host tissues have been postulated by different workers. Smith, believing the bacteria to be intra- as well as intercellular, described "tumor strands" by which the bacteria were carried for some distance through the stem of succulent plants by the proliferation of cells containing bacteria. One criticism of this theory is that the bacteria are not intracellular. Riker and others were of the opinion that the bacteria, first multiplying in the watery fluid of the ruptured cells, were carried inward by the movement of this intercellular fluid. Ivanoff and Riker got appearances like "zoogloeal strands" from dead bacteria and India ink, suggesting that various forces including capillarity and negative pressure were factors in migration. Hill and others have demonstrated the presence of zoogloeal strands and have measured the rate of migration in succulent plants. Hill recorded an advance of 5.25 mm in 3 hours. The rate of migration diminished with time, and judging from the rather superficial location of the bacteria in the woody plants, it must be much more limited in fruit trees and brambles. The tissues through which the zoogloeae penetrate are those in which the larger intercellular spaces occur, such as the subepidermal parenchyma.

The depth of penetration of the bacteria is also a matter of controversy. Riker and Keitt were able to isolate *Agrobacterium tumefaciens* from deep-seated tissue with ease, while Hildebrand in the case of *Agr. rhizogenes* found that the organism was confined to the soft parenchyma near the surface. The exact cause of the hypertrophy and hyperplasia produced in these malformations is not known. The stimulating material evidently acts a short distance from the bacteria and consequently must be soluble in the plant fluids, but that it is localized is evidenced by the fact that the enlargements occur only near the point of inoculation.

The bacteria of both crown gall and hairy root are widely distributed in the soil where susceptible plants are grown. Bacteria produced on or near the surface of the galls are washed off by the soil water in contact with the diseased tissue. Washings from crown gall or hairy roots have been shown to contain large quantities of the bacteria. Experiments have proved that they are able to survive the winter in the soil independent of the plant. Both crown-gall and hairy-root organisms may be disseminated on nursery stock which appears free from those diseases since they may be carried in soil particles, in incipient galls, or on the surface tissues.

Control. Control of hairy root and infectious crown gall has been greatly facilitated by the recent research which has cleared up many points in the life history of the organism. Furthermore these investigations have explained many of the reasons for wound overgrowths and demonstrated how these can be prevented.

So far as fruit trees are concerned, most infections result from the grafting process when the bacteria are introduced from various sources. It has been found that crown gall and hairy root are greatly reduced when budding, rather than grafting, is practiced.

The seedlings from which the rootstock is secured for piece grafting is often infected to a limited extent in the field. The seedlings are bunched and kept moist, thus giving a chance for the distribution of the bacteria over the surface of the healthy seedlings. Later when these are cut the bacteria are distributed over the surface of the wounds where they are in a favorable position for infection. For this reason the seedlings should be thoroughly washed and dipped in a disinfectant and dried before cutting. For the prevention of wound overgrowth and the protection of the graft union, a special adhesive tape has been developed. This should be used in preference to the string wrap.

Special attention should be given to fitting the grafts in such a manner as to prevent the tip or edge of the cion from projecting beyond the root piece. This prevents the type of gall known as wound overgrowth. Directions on this point have been printed in various publications for nurserymen.

Soil in which crops susceptible to crown gall have been recently grown should be avoided in planting out nursery stock. Corn and other grain crops should be planted for several years previous to replanting a nursery block. Soil in which root-feeding insects are present should be avoided if possible.

Regardless of whether or not overgrowths are infectious, the orchardist should insist on trees which have a smooth union at the graft and no evidence of hairy-root growth at this point. Apple or peach orchards should not be planted on land in which brambles showing abundant crown gall have recently been grown. It is safer to plant apples than peaches under such conditions. Since the crown gall organism enters only through comparatively fresh wounds, it is evident that care should be taken to avoid all unnecessary wounding of the roots and lower trunk of both nursery and orchard trees.

While some varieties of apple show less crown gall and hairy root than others in the nursery, it is not practical to control the disease by the use of resistant varieties.

Peach seedlings in the nursery rows often become infected through small wounds at the junction of the root and stem. These appear more frequently in alkaline than in acid soil. Nurserymen should avoid alkaline soil, especially where lime has been added in recent years. Good control has been obtained by a dust treatment of the washed seeds with ceresan or mercuric iodide dusts.

Crown gall has been "cured" by the application of certain antibiotics, but at present this is not practical.

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Apple Blister Canker

Blister canker is also commonly known as "nailhead" canker. It was called "Illinois apple tree canker" by Hasselbring when first described as a serious disease of the apple, in order to distinguish it from "New York apple tree canker," which was caused by the black rot fungus.

History. The fungus causing the disease was first mentioned as occurring on apple trees by Schweinitz as early as 1831, although it was not regarded as a serious disease of the apple until attention was called to the substantial losses in Illinois orchards by Hasselbring in 1902. The fungus causing blister canker was mentioned frequently between those dates from both Europe and America, but was always considered a saprophyte, and no mention is made of its being the cause of the death of fruit trees. Since the fungus was found generally distributed on wild hosts both in America and Europe, there is some question as to the original home of the disease.

As stated above, there seems to be a difference in the parasitic behavior of the fungus in different parts of the world. If one considers the distribution of the *fungus* it may be regarded as generally distributed in both North America and Europe, but if one considers the *disease*, especially on apple, it may be said to be prevalent in the apple-growing regions of eastern and central United States and especially destructive in the Mississippi Valley. The fact that the most susceptible variety of apple, Ben Davis, was for many years one of the principal varieties in this region may account for the prevalence of the disease in the central and Great Plains states. It has never been reported from west of the Rocky Mountains as an apple disease.

Economic Importance. The main loss from blister canker is due to early death of the tree or loss of fruit-bearing limbs. Often one or more of the main limbs are killed, and the exposure of other limbs to the hot sun results in severe sun scald, with consequent weakening and final death. On susceptible varieties, such as Ben Davis and Gano, 10 per cent or more of the bearing wood is unproductive. The greatest loss by far results from the early decline of Ben Davis plantings. An orchard of this variety soon becomes so badly diseased that it is no longer profitable to take care of the remaining straggling trees. The average productive life of a Ben Davis orchard is about 25 years, while orchards planted to nonsusceptible varieties are at their maximum period of production at this age.

In one county in Illinois during a 4-year period, 50,000 Ben Davis trees were removed between 1924 to 1929 largely because of blister canker. It is rare to find Ben Davis orchards over 35 years of age in the regions where this disease is prevalent, although 50-year-old trees of this variety bear excellent crops when not diseased. The susceptibility of this variety was responsible in part for its decline in popularity. In 1920, 37 per cent of the bearing trees in Illinois were of this variety, while by 1949 they had been reduced to 2.3 per cent.

Symptoms. There is no evidence of blister canker on the fruit, foliage, or young twig growth. The disease is to be looked for on the trunk and larger branches. It is most likely to be found around large pruning wounds. Typically the cankered area is elliptical or oval in outline. The surface of the diseased bark at first is smooth, but at the end of the first year of development, even when the fruiting bodies do not develop, there is a definite line between the healthy and diseased wood. The bark is killed to the cambium throughout the canker. This would be expected since the fungus grows from the wood out into the bark. The invaded bark ordinarily remains rather soft, and is never horny in texture, as is the case in black rot cankers. When dried, as in the center of old cankers, it breaks off easily in small chunks. The mottled appearance of the inner bark is the most characteristic diagnostic feature of blister canker in the absence of the fruiting bodies. This is most evident along the edge of the canker where the recent invasion of the fungus has occurred. Cream-colored irregular areas alternate with brownish areas (Fig. 28A) in such a way as to produce a mottled effect, which is quite different from the regular zonate appearance often seen in black rot cankers.

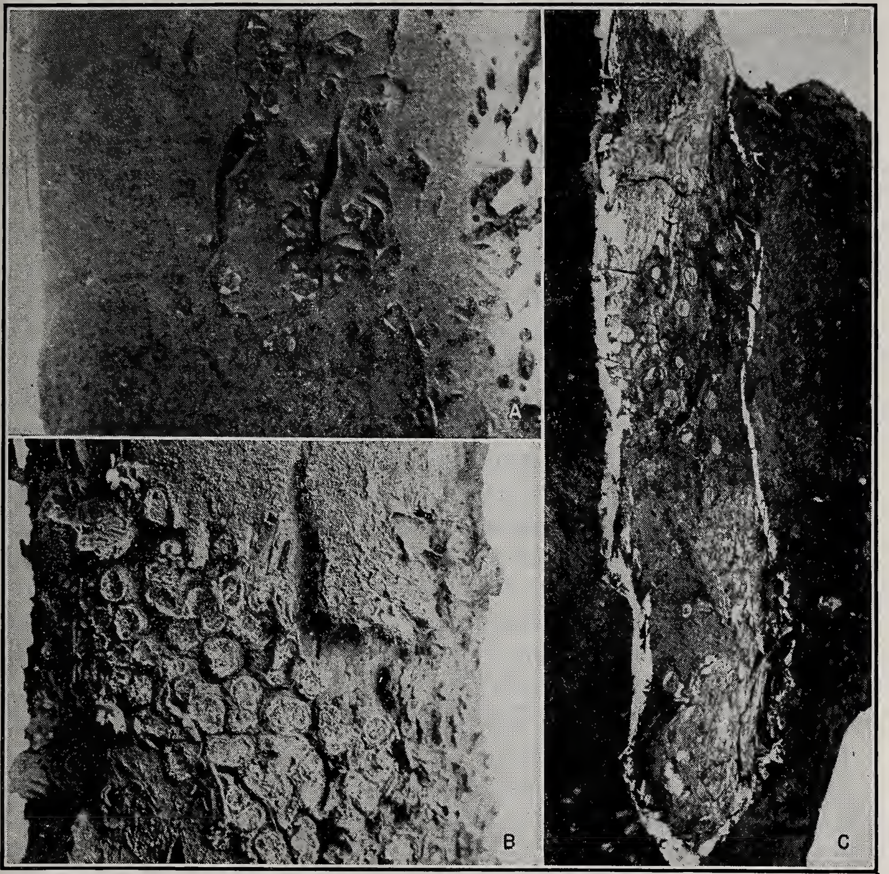

Fig. 28. Apple blister, or nailhead canker, on apple limb. A, rolled-back edges of blister of imperfect stage; B, nailheads of perfect stage; C, section exposing inner bark showing black rings of young blisters above and mottled bark below.

The smooth surface of the bark is later broken above by numerous small protuberances. The outermost layer of bark rolls back from the breaks, producing a star-shaped opening and exposing the honey-colored masses of spores which have formed beneath (Fig. 28A). At this time a cut exposing the inner bark will reveal black circles ⅛ to ⅜ in. in diameter. The surface of the canker from this time on becomes increasingly roughened by the breaking through of the fruiting structures known as "nailheads" (Fig. 28B). These consist of numerous circular disks which extend from the wood to slightly above the surface of the bark. Externally they are coal black in color, especially when weathered, and are usually from 1/4 to 3/8 in. in diameter. These disks are firmly attached to the wood and often remain after the more friable dead bark has worn away. When broken loose from the wood, the black ring remains in evidence for some years. The resemblance of these disks to the thick heads of old-fashioned iron nails gave rise to the common name of the disease.

While the edge of the canker is usually rather sharply defined, it is rare to find any callus formation. When the canker has progressed to the extent that the limb is girdled, the branch dies and the presence of these dead branches in what appears to be a healthy tree often calls attention to the disease in an orchard.

Cross sections and longisections of limbs of diseased trees show characteristic features in the wood. In a cross section of a normal limb, the heartwood is somewhat darker than the sapwood and the two are separated by a regular ring. When the blister canker fungus has invaded a limb, the affected wood is much darker and has a watery appearance. The area in cross section is irregular in outline, often with fan-shaped segments. At the lower or upper extremity of the infected area, dark brown lines may extend some distance beyond the generally invaded tissue.

Causal Organism. Blister canker is caused by one of the Ascomycetes, *Nummularia discreta* (Schw.) Tul.[1] It was originally described by Schweinitz in 1831 as *Sphaeria discreta.* The generic names *Diatrype* and *Hypoxylon* have been used.

Hosts. This fungus has been reported on rather widely separated host plants such as *Betula* (birch), *Magnolia,* elm, and *Gleditsia* (honey locust). Among the plants related to the apple it has been found on pear, serviceberry, and mountain ash. It is especially common on mountain ash wherever this tree is grown.

Varietal differences in susceptibility are marked. Undoubtedly Ben Davis and Gano are by far the most susceptible varieties, and it is rare to find an orchard of these varieties over 20 years old in which blister canker is not present. Yellow Transparent and Red Delicious are listed as being especially susceptible in some regions. The disease has been reported on all other common varieties with no marked difference in susceptibility. In general, it may be stated that those varieties which make a rapid growth with long, unbranched main limbs are more subject to blister canker than those making a slow, bushy type of growth.

Morphology. The nailheads of *Nummularia discreta* are the fruiting bodies of the fungus and are called *stromata.* When mature, they consist of a combination of bark and fungus tissue. The development of the stroma is as follows: After the

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[1] Class Ascomycetes, Order Sphaeriales, Family Xylariaceae.

bark is invaded, a mass of mycelium is formed just underneath a lenticel. This enlarges and forms a dark cushion, which by its enlargement pushed the periderm upward, thus forming the "blister." This lenticular mass is composed of sclerotial-like cells. In the center of this a flat chamber is formed by a horizontal splitting of the fungus tissue. On the walls of this chamber, short, sparsely branched *conidiophores* are formed, and on these the one-celled, hyaline, oval *conidia* are produced. This chamber ruptures and forms the star-shaped opening with the honey-colored masses of conidia previously described. The rolled-back segments of the bark and upper walls of the chamber carry the conidial masses so that they are exposed to the weather. From the outer edge of this disk the sclerotial growth extends inward to the wood and may grow into the outer ring of wood tissue and thus form a firm connection between the bark and wood. This outer layer of fungus tissue is black in color, so that a longisection of the stroma shows a black line extending from the top cushion into the wood. The interior of the stroma between the top cushion and the wood is composed of alternate dark and light layers of a mixture of bark cells and fungus mycelium. Ordinarily only a few of the stromata continue to develop, so that the number of nailheads may be much less on a given canker than the number of blisters formed. Sometimes the sclerotial cells do not extend beyond the cambium, and the nailheads are easily removed from wood. When attached to the wood the nailheads often remain for several years after the surrounding bark is worn away.

The *perithecia* are formed in the upper layer of sclerotial cells. This outer cushion thickens and forms a flat or slightly concaved disk underneath the old conidial chamber. Certain cells at the base of the sclerotial mass become active and form the perithecial chambers. A single stroma may contain 25 or more perithecia. The perithecia, when mature, are rather deeply imbedded in the stroma and are elongated flask-shaped, with a narrow neck extending slightly beyond the surface of the stroma. The neck varies in length according to the depth of the perithecial chamber, but it is usually from ¼ to ⅒ the length of the perithecium. The perithecium including the neck is from 1 to 1.5 mm in length. There is a distinct separation of the wall of the perithecium from the surrounding stromatal tissue, so that the entire perithecium may be lifted out of the chamber when it is mature. In a longisection of the stroma the perithecia, when wet, may be seen with the naked eye as whitish gelatinous bags suspended in a black-walled chamber.

The cylindrical, obtuse *asci* arise from the bottom and side walls of the perithecium. They taper at the lower end into a short stalk and are from 110 to 160 by 10 to 15 μ, averaging 140 by 12 μ. The uniseriate *ascospores* are dark in color, subglobose, averaging 11 by 13 μ. The ascospores darken with age, and when black a distinct hyaline zone can be observed on one face of the spore. The walls of the asci are easily ruptured, and it is rare to find the ascospores in the asci. The ascospores are forcibly discharged and may be carried some distance above the surface of the stroma. Frequently the surface of the stroma is covered with a layer of sclerotial tissue representing the old conidial chamber, and this must weather off before the ascospores are discharged into the open air. Dark specks observed on the surface of the stromal disk are masses of the black discharged ascospores.

Disease Cycle. Both the ascospores and conidia are capable of bringing about infection. The conidia, however, are short-lived and probably play little part in natural infection. The ascospores, on the other hand, have been known to be viable for 3 years after they were formed and are probably responsible for the majority of natural infections. Water, wind, birds, insects, and workmen are the agents probably responsible for the dissemination of the spores. Infection generally takes place through wounds which expose the older heartwood, since it has been demonstrated that wood less than 3 years old is not favorable for infection. The mycelium thrives best in relatively dry heartwood and when established makes its way in both directions from the point of inoculation. It may progress at the rate of from 1 to 3 in. per week. The infected area may extend throughout the tree before there is much evidence of canker production. The cankers are often formed by the *outward* growth of the fungus from the heartwood to the surface. It is common to see cankers developing around a large pruning wound, but this is not necessarily evidence that infection took place at this point. The fungus tends to grow more rapidly in the drier wood bordering on an exposed wound, and this accounts for the appearance of the cankered area about the wound. Cankers sometimes appear along an unbroken surface of a limb, in which case a cross section shows that the heartwood in this region is close to the surface.

There is a definite relation between the water content of the wood and the growth of the fungus. Following dry years, blister canker is likely to be severe.

While the invading mycelium is rarely active in healthy bark, it progresses rapidly in the bark outside the invaded wood, and thus a large cankered area may be produced during one growing season. It is not uncommon to see a 4-inch limb girdled in 3 or 4 years after the first appearance of the canker.

Little is known about the time of natural infection. Apparently ascospores can infect at any season of the year, although inoculations during spring and fall are more likely to produce infections. In most cases, infections may be traced to wounds in which deep cracks occur. This is probably due to the fact that moist conditions are maintained over a time sufficient to permit germination and the establishment of the mycelium in the wood. Inoculation of clean-cut surface wounds usually results in failure of infection. Infections in the orchard have been traced to frost cracks and to splintered stubs, which often result from the breaking off of overloaded branches.

Control. Control of blister canker is based on general orchard management, rather than on special control measures. Varieties such as Ben Davis and Gano which are especially susceptible to the disease should not be planted. The trees should be shaped early so that large pruning wounds in the older trees may be avoided. Blister canker is rarely found in trees less than 10 years of age, so that ample time is given to shape the trees properly for future growth. Where large limbs are removed in older orchards, the cuts should be made in such a way as to avoid cracking, and the wounds should be shellacked, or otherwise waterproofed, until they heal. Special attention should be given to broken limbs to see that no splintered stubs are left.

The appearance of a canker on a tree usually indicates that a more or less general infection of the wood of the cankered limb has occurred. The limb should be cut off some distance below the canker and the wood examined. If the characteristic brown areas of infected wood extend into the trunk, it is not possible to eliminate the disease. Since diseased trees may bear a number of valuable crops, the best procedure is to mark the tree and watch it for cankers in subsequent seasons.

When a number of trees in an orchard are known to be infected, the best procedure is to cut away the cankered bark to prevent the production of spores until the trees become so badly cankered as to be worthless, when they should be removed. Pruning tools, especially saws, should be disinfected or thoroughly washed after being used on a diseased tree.

Spraying for the control of other diseases probably reduces the amount of infection, but since blister canker often appears in orchards which are sprayed annually, it is evident that complete control is not secured.

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Phytophthora Collar Rot of Apple

This disease was known in the north central states for many years as "Grimes collar rot." After the discovery of the true cause by Baines it was called "*Phytophthora* trunk canker," which is more appropriate, since a number of other agents may bring about "collar rot." The "collar" location of the disease would warrant changing to the common name here given.

History. Collar rot as a cause of the dying of apple trees has long been known and often mentioned by horticulturists and growers. In most cases it is difficult to determine from the description whether these statements refer to the specific disease here treated. Some indication of the cause is suggested when varieties such as Grimes are mentioned. Collar rot resulting from winter injury also may be eliminated by taking into account the descriptions of the type of injury and the recorded weather conditions in the area concerned. Collar rot of Grimes in particular was mentioned as early as 1858. The extensive planting of Grimes in the upper north central states during the last half of the past century soon called attention to this weakness, and collar rot of Grimes soon became a common complaint of the orchardists. Selby in Ohio called particular attention in 1900 to a collar rot which he ascribed to winter injury, but mentioned that Grimes trees were especially affected. Burton in Indiana the same year spoke of "Grimes bark disease which affects it at the ground or a little above." It seemed to be generally realized that there were some differences between the type of injury caused by low temperatures and the type observed on Grimes. Often 10 to 15 per cent of Grimes would develop collar rot in an orchard where winter weather conditions has been such as to eliminate this agent as a cause. Furthermore, when winter injury occurred it was usually found to affect trees on one side only, usually the south or southwest side, while on the Grimes no such uniform location was observed. The failure of fruit pathologists in most cases to isolate a causal organism, however, supported the theory that the cause might be weather conditions or an inherent weakness in the trunk of the Grimes variety.

It was well known that collar rot could be caused by the fire blight organism on both apple and pear, but the failure in most cases to recover this organism from young lesions on Grimes discounted this agent, although Orton and Adams in 1915 in Pennsylvania reported consistent isolations from what was described as typical collar rot and mentioned Grimes as one of the susceptible varieties.

In 1939 Baines published the results of an extensive investigation of the cause of collar rot in which he pointed out some of the reasons for the failure of plant pathologists to isolate the organism concerned. His work also showed why Grimes had so often been recorded as suffering from the disease. He found that a soil-inhabiting Phycomycete identified as *Phytophthora cactorum* was responsible and that Grimes was by far the most susceptible variety, as demonstrated by numerous inoculations. This organism is known to cause a fairly common fruit rot and had previously been known to attack a large number of plants causing various types of injury. This fungus is widely distributed and very common in orchard soils. The very thorough investigations of Baines answered many questions, such as why this disease was especially prevalent on Grimes, why it appeared most frequently at the ground line, and why double working of Grimes was often effective in control, a fact which had long been known.

Geographic Distribution. The fungus causing collar rot may be said to be universally present in soils capable of sustaining its growth. As a collar rot of apple the disease is probably limited to where the very susceptible varieties such as Grimes are commonly grown and where soil conditions are favorable for its sustained growth. Very few of the standard varieties of apples are susceptible, and consequently little loss of trees is sustained. The fruit rot is more widespread.

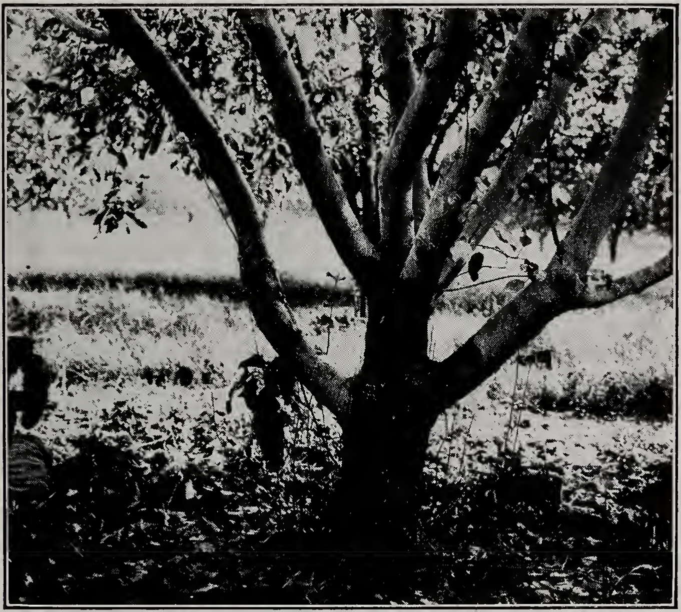

Fig. 29. *Phytophthora* collar rot of apple on Grimes. Advanced stage with extension to base of main branches. (*Photograph by R. C. Bains.*)

Symptoms. While primarily a trunk disease, cankers occasionally appear in crotches of the scaffold branches. Usually, however, the cankered area is near the ground line and appears most frequently on trees 5 years or older. Young cankers are hard to detect, but after the killing of the bark a moist exudate may be noticed over the invaded area. In this stage the inner bark is slimy. The canker is irregular in outline but usually roughly oval. Rapid extension of the cankered area both in a lateral and vertical direction occurs, often resulting in girdling the tree in one season, but ordinarily confined the first season to one side of the trunk (Fig. 29). In old cankers the bark dries out and becomes more definite in outline.

If the tree survives, a callus is formed along some of the peripheral area, but this may not completely delimit the canker since streaks and lobes of dead tissue may extend into the healthy bark tissues. Usually the cankered area extends downward only to the graft union, since the stock on which the Grimes is grafted is resistant. On own-rooted Grimes the canker may extend into the main roots.

Trees attacked by this disease may be recognized at a distance by the lighter foliage. This light-colored foliage often appears only on the branches above the cankered area, while the remainder of the tree may appear normal and may bear a crop for several years.

Examination of the cankers the year following infection will show an extension of the fungus in streaks or broader lobes. In some cases the fungus dies out and the callus growth heals over the entire wound.

Economic Importance. The loss from *Phytophthora* collar rot may be extensive on Grimes and Tompkins King. Other varieties are rarely attacked. The fact that trees near their maximum bearing period are especially susceptible must be taken into consideration in estimating losses. The grower has gone to the expense of bringing the orchard into bearing and perhaps harvests two or three crops, and then over a period of a few years 50 per cent or more of the trees are killed.

Baines reported that in Indiana, following severe outbreaks in 1933 and 1934, a survey of six orchards 14 to 18 years old showed 21 to 68 per cent of the trees infected. In Illinois an 18-year-old orchard of Grimes had 72 per cent of the trees infected or missing (removed following death from collar rot). Thus Grimes orchards may be almost entirely destroyed at a period when the next few years would be the most productive.

Fruit rot caused by the collar rot organism is fairly common but is of minor importance in most seasons.

Causal Organism. Collar rot is caused by *Phytophthora cactorum* (Lebert.-Cohn) Schröt.,[1] a fungus, belonging to the "downy mildew" group of the Phycomycetes. As stated by Fitzpatrick, "A satisfactory basis for separation of species within the genus *Phytophthora* has not yet been found." For this reason only a few of the morphological details of this fungus will be given.

The nonseptate *mycelium* gives rise to sympodial branched *conidiophores* with the production of lemon-shaped *sporangia*. Within the sporangium, which is usually 50 μ or more in length, from 40 to 90 *zoospores* are produced. These zoospores are typical of the genus, with two lateral cilia. They germinate by a single germ tube shortly after they are released from the sporangium. The sexual stage is also typical of the group. *Oöspores* have been found in abundance in the younger portion of the cankered bark. These probably function as resting spores.

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[1] Class Phycomycetes, Order Peronosporales, Family Pythiaceae.

They germinate by means of a germ tube which may soon produce a conidiophore or may develop an extensive mycelial growth in the host or in the soil.

Biologic Strains. It was demonstrated by Baines that the composite species, *Phytophthora cactorum,* contains a number of distinct strains having different potentialities for infection. Cultures of this fungus originally isolated from nine different host plants failed to infect Grimes. One culture obtained from a lily in Indiana did infect Tompkins King, Rome Beauty, and Stark. Most of these isolations as well as the apple strain were able to infect peony and flax.

Disease Cycle. Little is known concerning the source of initial infection of the apple tree. It has been found that mycelial infection can readily be brought about by inserting diseased bark or cultures of the fungus into the bark of the trunk or larger limbs of susceptible varieties. Application of damp infested soil will also result in infection. Since the initial infection is usually near the ground line and since the fungus is known to be present in orchard soils, it is probable that the majority of infections occur through mycelial penetration. The fungus rarely produces conidia (sporangia) on the surface of the cankers, but zoospore infection is not ruled out since these may be liberated in the soil or on rotten apples. Some fruit rot occurs practically every season in a bearing Grimes orchard. Pieces of bark containing the oöspores might also be a source of infection. Infection above the ground line or in tree crotches probably results from infested soil or debris deposited by splashing or wind-blown dust. It is well known that most of the apple-fruit infection is found on the lower branches, where they are subject to splashing.

Infection usually occurs during damp cool weather in the spring, although the cankers are hard to detect until later in the season. Penetration apparently can take place through unwounded bark, but it is probable that in most cases cracks or small wounds serve as infection courts. The fungus grows both intra- and intercellularly through the bark tissues, which are soon killed. The tree may be girdled within 3 or 4 months. Penetration slows down toward fall, and on trees not girdled a callus is formed around the edge of the canker. In many cases where the tree is not girdled the fungus continues invasion of the live tissue the following spring. A few cankers fail to develop further in the second or subsequent years. Secondary infection from existing cankers is probably of no importance, if it occurs at all. The fungus is able to survive indefinitely in the orchard soils either as mycelium or through oöspore formation.

Varietal Susceptibility and Relation of Age of Tree to Infection. For many years growers recognized the fact that relatively few varieties of apple were affected by this collar rot. Two varieties, Grimes and Tompkins King, were recognized as especially susceptible. The inoculations made by Baines of a number of varieties proved that this varietal susceptibility was very marked. Of 34 varieties trunk-inoculated, only 6 developed cankers. While limited in extent, these experiments supported the results of numerous orchard surveys in which it was only rarely that this type of crown rot occurred on varieties other than Grimes and Tompkins King. The fruit of a large number of varieties, however, is susceptible.

Under natural conditions cankers can rarely be found on Grimes trees under 8 years of age. Orchardists usually mention the period between 10 and 15 years as the time when trees "began giving out." Again Baines showed that these observations were based on facts. It was difficult to infect Grimes trees under 5 years of age, while high positive infections were obtained on trees 10 years or older. Also younger branches on these older trees resisted infection. The reason for the resistance of younger trees has not been explained.

Control. "Double working" of Grimes trees was advocated many years before the true cause of collar rot was known. This consisted of grafting a cion of a resistant variety on the seedling stock and a second grafting or budding of the Grimes 12 to 15 in. above the first graft. In some cases top grafting of established varieties was resorted to by the growers. In the opinion of many growers the use of double-worked trees materially reduced the loss of trees. However, Baines in a survey of a large orchard of double-worked Grimes found a high percentage of cankers even on trees with the graft union 15 or more inches above the soil line. This survey was made following 2 years of epidemic conditions and does not rule out the benefit obtained by double working, especially if the Grimes cion is inserted high on the intermediate cion.

Grimes and Tompkins King should be avoided in planning a new orchard or in replanting where trees in a bearing orchard have been removed. In a bearing orchard, trees which have not been killed may be saved by prompt removal of the diseased bark and bridge grafting with the lower end of the graft stick inserted below the graft union.

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Apple Anthracnose

This disease has been referred to as "northwestern anthracnose," "black spot canker," "Pacific coast canker," and "apple tree anthracnose."

History and Geographic Distribution. Attention was first called to this disease in the Pacific northwest in the early 1890s, and Pierce, a pathologist in the U.S. Department of Agriculture, was sent to this region to investigate the malady. His results were not published, but he established the fact that it was of parasitic origin and that it was confined to rather definite areas in the states of Washington and Oregon. In 1900 Cordley published the results of an investigation of the disease in Oregon and described and named the causal fungus. Lawrence in Washington confirmed the results obtained by Cordley and connected a storage fruit rot with the canker phase of the disease. Jackson in 1913 reported his discovery of the perfect stage of the fungus, which had hitherto been known in the imperfect, or *Gloeosporium*, form. In 1925 a similar canker disease of apple called "perennial canker" was discovered and studied by Zeller and Childs. This occurred in the drier regions of the Northwest, but in many respects resembled anthracnose. Keinholz made a detailed comparison of the two fungi causing these diseases and reported in 1939 that intraspecific variations in the two species were as great as interspecific differences. He found the perfect stage of the perennial canker fungus, which also corresponded closely with the ascigerous stage of the anthracnose fungus.

Apple anthracnose is almost exclusively confined to the humid coastal regions of Oregon, Washington, and British Columbia, although it has been reported from Maine, Illinois, Massachusetts, and Oklahoma, but is regarded as of little importance in these states. It probably occurred on some wild host in the Pacific Coast region and went to the apple when extensive orchards were planted in this region. The fact that it is limited in its distribution is evidently due to climatic relations. The fungus causing the disease has been reported from New Zealand, Denmark, and Holland.

Symptoms. Cankers are formed on the *younger branches*, usually those less than 2 in. in diameter. These cankers are elliptical, varying in size from 1 in. to 10 in. in length and 2 or 3 in. in diameter. The surface of the sunken lesions in the fall shows numerous transverse slits. The mature cankers have a definite crack in the bark, delimiting the dead central area. The diseased bark in old cankers wears away, exposing wood. A callus layer forms, making a pronounced ridge around the edge of the wound. The cankers along a limb may be scattered or very numerous. The first evidence of new infections is the appearance of small reddish-brown spots with discolored bark extending inward to the cambium. Such spots may be observed in the late fall, and by midspring of the following year these reach full size, but the surface is smooth until midsummer, when numerous dark pustules appear which later break through to the surface, exposing the cream-colored spore masses.

On the *fruit* the rot resembles bitter rot lesions, but is more depressed and enlarges more slowly. The tissue is spongy beneath the brown epidermis. Anthracnose on the fruit usually appears late in the season and often does not make its appearance until the fruit is harvested or in storage.

Economic Importance. The limited distribution of the disease accounts for the minor loss to the apple-producing industry as a whole. The cankers are most destructive to young trees where the branches may be girdled and killed by numerous cankers or the trees so weakened as to reduce production materially when they come into bearing.

The fruit rot may cause an almost total loss of fruit, although, like bitter rot, it is sporadic in its appearance and severity.

Causal Organism. The fungus responsible for apple anthracnose is *Neofabraea malicorticis* (Cord.) Jack.[1] The imperfect stage is a *Gloeosporium* described by Cordley in 1900 and named *G. malicorticis.* In the same year Peck named the fungus *Macrosporium curvispora* from a specimen from British Columbia. In 1909 Jackson found the ascigerous stage in 2-year-old cankers and described and named it in 1913. Cross inoculations by Lawrence proved that the fruit rot was caused by the same fungus as that responsible for the cankers.

Hosts. In the northwest region where anthracnose occurs, apple, pear, and quince are attacked. Inoculation experiments produced typical cankers on Oregon crab (*Malus rivularis*), serviceberry (*Amelanchier*), and mountain ash (*Sorbus* spp.). It is thought that the Oregon crab may have been the native host of this fungus since it is common throughout the region where the disease is most prevalent.

Morphology. The pustules (*acervuli*) scattered over the surface of the canker become erumpent and expose the mass of cream-colored spores which are borne on simple or branched conidiophores. The continuous, hyaline *spores* are characteristically curved or hooked, but according to Keinholz vary greatly in that under some conditions they may be almost straight. Variation in size is marked, but they are usually within the range of 3 to 6 by 15 to 20 μ. Microconidia 2 to 10 μ long are commonly found in the pustules and in culture.

The *apothecia* develop within the old conidial stromata during the year following conidial production. The *asci* are clavate, 90 to 100 by 10 to 13 μ, and in the apothecium are interspersed with simple or branched paraphyses. The *ascospores* are hyaline, continuous, and ellipsoid in shape, but often with a flattened side. They measure from 6 to 7 by 17 to 20 μ. The ascospores are forcibly discharged after the elongation of the ascus. The asci develop progressively so that spore discharge may occur at favorable intervals over a fairly long period. With age the apothecium turns from a gray or rose color to a deep brown.

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[1] Class Ascomycetes, Order Pezizales, Family Mollisiaceae.

Disease Cycle. Infection generally occurs during the fall, at which time the conidial pustules are fully developed and the conidia are exposed. Since the conidia are produced in a gelatinous matrix, it is probable that these are disseminated by meteoric water. Ascospores are forcibly discharged and may be carried by wind currents and thus promote wider dissemination of the inoculum. Infection may be through wounds or lenticels. The cankers start development in late fall as small, circular, reddish spots, but do not enlarge until the following spring. They reach full size in May, and during the summer the acervulus is organized in the tissues of the canker and the pustules erupt in the fall, that is, a year after infection occurs. The mycelium grows throughout the bark tissues and penetrates to the cambium and may penetrate the sapwood to a limited extent. At the end of the first summer the canker has reached full size, but the fungus remains alive in the dead bark of the canker. The following year the conidial stromata function as apothecia, producing the asci in the same pustules. The ascospores are discharged in the fall of the second year following infection. Infection of the fruit evidently occurs in the fall when both conidia and ascospores are being disseminated. Often no evidence of fruit infection is seen when the apples are harvested, but appears in storage. Infection is mainly through the lenticels on the fruit.

It is evident that anthracnose is greatly influenced by environmental conditions, since it is known to be severe only in those regions having frequent rains and moderate temperatures at the time of the production and dissemination of the spores.

Control. Anthracnose is far more prevalent in neglected orchards than those well cared for, thus indicating general sanitary measures are of importance. Varietal resistance is not marked, although it has been stated that Baldwin is especially susceptible, while Ben Davis and Northern Spy are seldom attacked.

Excision of cankers or cutting out of diseased limbs eliminates sources of infection. If this is practiced, the dead bark and prunings should be destroyed, since the fungus will continue to produce spores which may result in renewed infections.

Spraying. Bordeaux mixture (4-4-100) should be applied before the fruit is harvested in order to protect it from subsequent infection. Tree infections can be prevented by repeating the sprays (1) as soon as possible after harvesting and (2) about 2 weeks later.

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European Canker of Pome Fruits

History and Geographic Distribution. European canker is one of the most important diseases of apples and pears in Continental Europe and the British Isles, where it has long been known. In North America it is of importance along the northern Pacific Coast region and in northeastern United States and eastern Canada, but it occurs across the continent in northern United States and southern Canada. It is reported to cause serious damage to apple and pear in Chili in South America, South Africa, New Zealand, and Japan.

The early history of the disease is not clear, since at one time no distinction was made between "canker" in forest trees, caused in most cases by a closely related *Nectria*, and the true fruit tree canker organism. Since the true European canker also occurs on forest trees, it is impossible to determine which of the two were referred to in the early descriptions. It was certainly known in Europe as an apple tree canker before 1866. Between 1880 and the early part of the nineteenth century it was studied extensively in Germany, especially as it related to forest trees. Bresadola in 1901 gave specific rank to the *Nectria* on apple, which is named *Nectria galligena*, thus separating it from the commonly occurring forest tree canker generally caused by *N. ditissima*. Wiltshire in England initiated an extensive investigation of the disease between 1914 and 1925, in which he described the stem infection through leaf scars and scab lesions on the twigs, as well as through other types of wounds. Crowdy, working at the Long Ashton Experiment Station in England, has contributed to a more complete understanding of the mode of penetration, physiology, and seasonal behavior of the fungus.

The disease was not known in North America until it was recorded by Paddock in 1899 as occurring on apples in Nova Scotia and New York. In 1909 Brooks reported it as occurring in New Hampshire, and since then it has been recorded from various parts of northern United States, but was regarded as a minor disease in most cases.

European canker was considered primarily an apple disease in the East, but was first reported in the Pacific Coast region as a serious disease of certain pear varieties. About 1920 Zeller started an extensive investigation of the disease in Oregon, the results of which were recorded in detail in 1926. He was of the opinion that the disease on both apples and pears had been present in this region at least as early as 1915. He studied the relation of the development of the disease to climatic factors, soil conditions, types of infection courts, age and vigor of trees, and varieties.

Economic Importance. In Europe, where in many instances it has been reported as causing the death of thousands of apple trees, this canker is considered of paramount importance. In addition it has a crippling effect on the bearing trees where limbs are girdled or reduced in vigor. Some especially susceptible varieties are no longer grown, although otherwise considered desirable. In Chili, Castillo reports it as extremely destructive in certain areas where climatic conditions are favorable for its development. Losses during the first 15 years of the life of the tree are more extensive than later. This is in part due to the fact that the fungus often girdles the trunk or scaffold branches during this period, while in older trees only small branches may be lost.

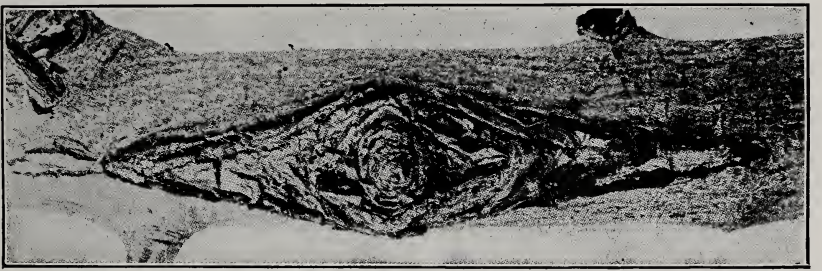

Fig. 30. European canker on pear, originating at broken twig. (*Ore. Agr. Expt. Sta.*)

With the exception of certain regions along the Pacific Coast, the disease is considered of minor importance in North America.

Symptoms. In an advanced stage it is not difficult to distinguish this canker from other types of canker on apple and pear. Removal of adhering flakes of bark reveals a series of callus folds surrounding a central cavity which extends to the wood. These cankers are usually centered around bud scars, wounds, twig stubs, or in the crotches of the limbs (Fig. 30). The folds are black and may number from one to several, depending on the age of the canker. Except in the case of the crotch cankers they are elliptical in outline and vary in length from 2 to 15 in. The younger infections are more difficult to detect, since during the first year they are covered and not easily distinguished from those caused by other agents. They start as small circular brown areas, usually around a leaf scar or superficial wound. Later the central area becomes sunken, with the edges raised above the surrounding healthy bark. Ordinarily the outward growth of the fungus is slow and is limited by a peripheral crack in the bark. Removal of the dry outer bark reveals dead spongy tissue extending to the wood. The dry, black outer bark cracks and flakes off, and the dry, spongy inner bark falls out, revealing the callus fold around the cavity.

While the above symptoms are typical of the cankers as usually observed under normal climatic conditions, it has been pointed out by a number of workers that another type, the "closed canker," is sometimes even more prevalent than the "open" type. This is especially the case where the canker originates in winter-injured areas of the bark or where climatic conditions are more favorable for the invasion of the fungus beyond the infection court. In the open type the predominance of the host-tissue growth prevents a rapid invasion beyond the first callus layer, so that the successive folds are in a narrow band around the cankered tissue. The closed type results from the rapid invasion of the host tissue during the second and successive period of active growth of the fungus, so that the successive concentric rings of callus are formed some distance apart. The bark above the invaded area is roughened and cracked, but often remains intact for several years. All gradations may be found between these two symptom types.

Along the margin of the canker the brilliant red globular fruiting bodies of the pathogen may be observed, but these are sometimes not formed or are scarce and scattered. Sporodochia giving rise to masses of conidia also may be observed on the surface of the folds. A fruit rot caused by this fungus has been reported, but is rare. A dark brown sunken spot results.

The callus tissue in the cankers is often covered with woolly apple aphids, and in some cases the feeding of this insect in a wound may bring about a condition very similar to that produced by the European canker fungus.

Causal Organism. European canker is incited by an Ascomycete, *Nectria galligena* Bres.[1] The imperfect stage is *Cylindrosporium mali*, but was originally described as *Fusarium mali*.

The bright red *perithecia* are formed on the surface of the cankered area or on detached limbs where the fungus is growing saprophytically. They are produced singly and may be scattered or closely crowded in the crevices of the bark. In some cases they seem to arise from the sporodochia or from a mat of interwoven mycelium, but not from a distinct stroma. They are broadly ovate, measuring about 0.5 mm in height and 0.3 to 0.4 mm in diameter. The *asci* are cylindric and distinctly stalked, measuring 90 to 125 by 8 to 15 μ. Very numerous narrow paraphyses are present. The *ascospores* are hyaline, distinctly two-celled, smooth walled, and usually elliptical in shape, 14 to 21 by 5 to 7 μ. Two kinds of conidia are present in the imperfect stage, macro- and microconidia. These are produced on branched conidiophores arising from cream-colored or slightly yellowish sporodochia. The *macroconidia*, creamy yellow in mass, are cylindric, hyaline, three- to five-septate when mature. (Castillo in Chili reports two strains, one with one- to three-septate macroconidia, the other with three to five septa.) The macroconidia measure 52 to 64 by 4.5 to 5.5 μ, according to Zeller. The *microconidia* are produced by abstriction of the tips of hyphal branches. They are single-celled, hyaline, and measure 4 to 7 by 1 to 2 μ and so far as known are functionless.

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[1] Class Ascomycetes, Order Hypocreales, Family Hypocreaceae.

Hosts. *Nectria galligena* has been reported as parasitic on a number of forest trees, such as beech, willow, and poplar, as well as on pome and stone fruits. *N. ditissima* also causes a similar canker, especially on forest trees, but cross inoculations with *N. galligena* have proved that it will attack a rather wide range of forest trees. It is of most economic importance on apple and pear, but has been reported as causing some damage to stone fruits.

Disease Cycle. Perithecia appear in the cankers in the fall, and ascospores are mature by late fall or early winter. These are forcibly discharged or ooze from the ostiole when the bark is in a moist condition. Discharge continues throughout the spring months. Macroconidia also are formed during this period. The agents of dissemination of the spores may be insects, wind, or water. Woolly apple aphids and ants have been found to carry numerous ascospores on their bodies. Forcibly discharged ascospores are carried by wind currents, while macroconidia and oozing ascospores are probably disseminated by washing during rainy periods. Leaf scars are regarded by Wiltshire and Zeller as the main infection courts. With the drying out of the surface of these scars after leaf fall, numerous cracks appear in which the spores find lodgment. Here they germinate and gain entrance to the tissue surrounding the leaf scar and bud. Other points of invasion are pruning cuts, insect wounds, crotch and frost cracks, cankers caused by other fungi, and winter-injured tissue. During the fall the fungus penetrates to the cambium and into the wood. It extends outward through the cells of the cambium layer, cortex, and wood parenchyma, often without showing any external evidence. Renewed growth of host tissue in the spring stops the advance by the formation around the margin of the canker of a callus derived from cambium and cork cambium. New bark forms over the edge of the callus so that a swollen ridge results. The fungus now renews its penetration beyond the callus, thus enlarging the canker. Another callus is formed concentrically around the last. This continues for several seasons, resulting finally in the multiple folds observed in the older cankers of the open type. Somewhat the same process takes place in the formation of the closed type of canker, but the more rapid penetration by the fungus, especially in the region of the cambium, widens the area between the successive callus layers. New infection also occurs during the spring months from ascospores discharged early in the spring. New crops of perithecia and sporodochia are formed each year, and thus the disease cycle is completed.

Relation of Climatic and Other Factors. Regions where the European canker is a serious problem are characterized by relatively mild fall and winter temperature and heavy fall and spring precipitation or high humidity. Such climatic conditions favor the late-fall and early-spring infections and the growth of the invading fungus during the relatively mild early winter months while the trees are in a dormant condition and in the spring before growth starts.

In England it is reported that cankers are always worse on a low site and a clay subsoil. Zeller in Oregon states that "orchards grown on hillsides in lighter soils have not been subject to canker as much as those on heavier soil in lower places where air drainage is poor."

Vigorously growing trees appear to be more susceptible to canker than less vigorous ones. While older trees are susceptible, the greatest loss is sustained on trees up to 12 to 15 years of age. This is probably related to the greater vigor of the younger trees. It was demonstrated in England that the application of manure or high nitrogenous fertilizers greatly increased the number of cankers as contrasted to unfertilized trees.

Some varieties of apple are especially susceptible to the disease, but none are known to be highly resistant. In England, Worcester Permain, Ribston, Coxes Orange Pippin, and Wellington are regarded as most subject to attack, while in Oregon the disease is found most often on Northern Spy and Winter Banana. Anjou and Bosc are among the most susceptible pears. In Germany, Hubbardston and Canada Reinette were mentioned as susceptible, while Gravenstein was resistant. No explanation has been offered to account for varietal differences in susceptibility, but one might suggest that other factors such as type of growth, rather than inherent resistance, might be involved.

Control. In regions where this disease is of minor importance, no special control measures aside from regular spraying are necessary. Where the disease is of considerable importance, sanitary measures, excision of canker, removal of diseased branches, and special fall sprays are recommended.

Since the fungus lives and fruits for some time on dead wood, all prunings in the orchard should be destroyed. If the cankers occur on small limbs, these should be cut out during the regular pruning. Trunk and scaffold limb cankers should be cleaned out and cut well back to sound, healthy bark. These may be treated with a Bordeaux mixture paste.

Since most infections occur through the leaf scars in the fall, a good fungicidal spray such as a 8-8-100 Bordeaux mixture spray should be applied immediately after leaf fall.

Trees should not be overfertilized, especially during the first few years of growth. Varieties subject to winter injury should be avoided.

Interesting results have been obtained by treating cankers with growth-promoting chemicals, but it is doubtful if this is a practical method of control.

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Armillaria Root Rot

This root disease is also known as "mushroom root rot," "shoestring root rot," "crown rot," and "oak fungus." The fungus causing the disease is known to occur on a large number of hosts, including all fruit crops, but since it has received most attention in its relation to apple orchards, it will be treated under pome fruits. In Europe, it has received more attention as a forest-tree disease, especially on conifers.

History and Geographic Distribution. The fungus responsible for root rot was described as early as 1777, but was not recognized as a root parasite until the work of Hartig in Germany (1873–1874). He established the relationship between the rhizomorphs, known as *Rhizomorpha subterranea,* and the "toadstools," then called *Agaricus melleus.* Hartig worked mainly with forest trees, but mentioned that the fungus was parasitic on plum tree roots. In 1886, the root rot of grape was attributed to this fungus in Europe, and the next year was observed on this host in America. During the early part of the present century this disease received attention in the Mississippi Valley as a serious root rot of apple trees. At that time it was found especially common on land cleared of oak for the planting of apple orchards and was commonly known as the "oak fungus." The importance of the root rot declined in this section as the orchards became established on land free of remnants of forest trees. On the Pacific Coast, however, the increasing severity of the root rot is attested to by the number of publications between 1920 and 1950. Piper and Fletcher called attention to the seriousness of the disease on prunes. Lawrence (1910) studied this root rot in the Puget Sound region and made the first extensive observations regarding injury to brambles. It was reported by Horne in 1912 as a destructive disease of citrus fruits in California, and in later papers he discussed the distribution of the organism in the soil and its relation to the root system. The parasitism of the fungus and the method of penetration were studied in detail by Day (1927) on coniferous plants, and in 1934 Thomas gave a comprehensive review of all previous work on this subject and reported in detail a series of experiments on the exact method of penetration of the fungus, demonstrating beyond question that penetration normally occurs through the unwounded bark.

In Europe, most of the investigations had to do with forest tree root rot. In 1937, Van Vloten, in Holland, reported the presence of a number of cultural "strains" of the root rot fungus, and his results confirmed those of Thomas that penetration is only by means of rhizomorphs and never by single hyphae.

Armillaria root rot has been reported from all sections of the world. In America it seems to be more prevalent in the regions west of the Rocky Mountains, but has been reported from all other areas.

Economic Importance. While *Armillaria* root rot is not of great economic importance to the fruit industry as a whole, except in the Pacific Coast area, the loss of a few trees in the orchards where the disease occurs creates an unfavorable production problem. When a few trees are infected, the problem of confining the disease to a localized area has to be met. Often orchards are planted on recently cleared land by growers unfamiliar with the risk of root infection, and it is only after the trees have become established that the seriousness of the situation is realized. Frequently, areas in the orchard have to be abandoned because of the disease.

Symptoms. The aboveground symptoms are not different from those of many other root injuries and, consequently, are of little value from a diagnostic standpoint, although they do indicate root trouble and thus lead to a further examination. Trees suffering from this disease show a weakened condition of foliage and twig growth. Older trees may show evidence of injury on one or more limbs on one side, while young trees may be killed outright. Such trees should be examined for evidence of crown or root injury. *Armillaria* root rot is indicated by the presence of dead areas in the bark, on and around which dark brown or black threads (rhizomorphs) of the fungus may be discovered by careful examination. These rhizomorphs, commonly called "shoestring," are about the size of the lead of a pencil or somewhat smaller. They may be mistaken for dried rootlets when first observed, but are darker in color. Often they form a branched system over the root surface, with some strands penetrating the decayed bark and some extending into the surrounding soil (Fig. 31). White, fan-shaped mycelial mats may be found between the bark and the wood, but these structures are often present when the death of the root is due to other causes. The presence of the rhizomorphs is definite evidence of the true nature of the disease, since no other root rotting fungus produces these strands, and this fungus is not able to infect in the absence of rhizomorphs. Honey-colored groups of toadstools around the base of the tree are final evidence of the disease, but these often do not appear until the disease has destroyed most of the roots, and sometimes not at all.

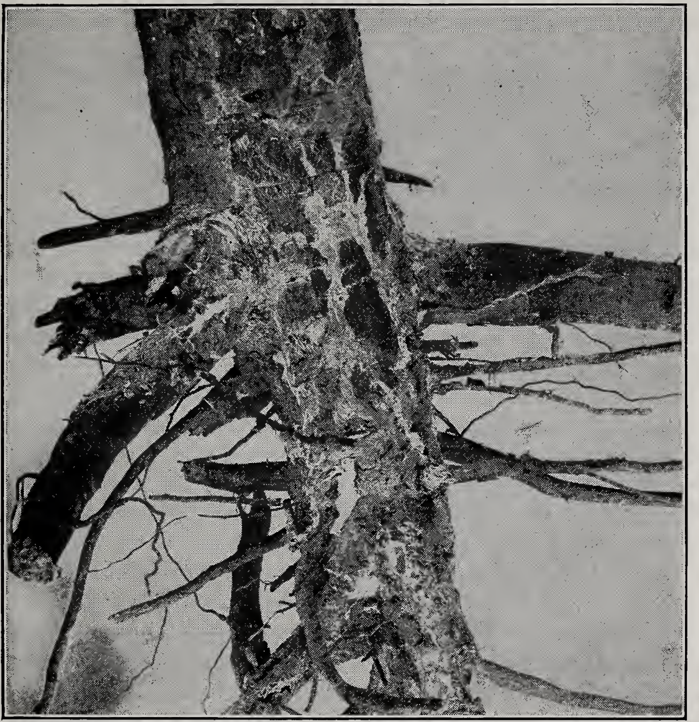

FIG. 31. *Armillaria* root rot showing strands extending along root at right.

Causal Organism. The fungus causing mushroom root rot, *Armillaria mellea* (Vahl) Quel.,[1] was first known only in the rhizomorphic stage and was named *Rhizomorpha subterranea.* The sporophores were described as

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[1] Class Basidiomycetes, Order Hymeniales, Family Agaricaceae.

Agaricus melleus, by Vahl, in 1777. The connection between the two was established by Robert Hartig, in 1874. The species was later transferred to the genus *Armillaria*. Some mycologists have discarded the genus *Armillaria* and have placed this species under *Clitocybe* or *Lepiota*.

Hosts. *Armillaria mellea* occurs on a large number of host plants, including not only woody but also herbaceous plants, such as potato. It is common on various species of forest trees and is perhaps most generally considered a forest-tree disease. It is also regarded as a serious parasite on citrus fruits and various shrubs, such as *Azalea* and *Rhododendron*.

Morphology. *Armillaria mellea* is a Basidiomycete, producing a typical gill-bearing "toadstool" sporophore. The sporophores are borne in clusters about the base of trees or stumps, the roots of which support the fungus in its parasitic stage. They are usually produced in the fall during the months of September to December. The stalk, or *stipe*, is ordinarily from 3 to 5 in. long. The convex cap, or *pileus*, is honey-colored above, with scattered brown scales, while the gills underneath are white. An *annulus* is often, but not always, present. This consists of a membranous ring below the gills. As in other fungi of this group, the gills are covered by a layer of spore-bearing, club-shaped bodies called *basidia*. The whole fruiting surface is known as the *hymenium*. From the outer, blunt end of each of these basidia, four narrow stalks arise, the stigmata, on each of which is borne a hyaline, elliptical basidiospore, 6 by 9 μ. These spores are forcibly discharged and fall down between the gills. There is some question as to the importance of the basidiospores in the dissemination of the fungus. It is probable that most root infections arise from the mycelium which has existed for many years in the soil on decaying roots in the vegetative state and that the amount of infection in an orchard is not related to the production of sporophores.

The *rhizomorphs* are composed of interwoven hyphae. The tips of the rhizomorphs are white in color, but when exposed or growing on the surface of the root, they are dark gray or black. They are often shiny, because of the production of a gelatinous material which aids in attaching them to the root surface. When growing in the interior of the bark, the rhizomorphs may remain white. These structures are very important in the life history of the fungus, since they are responsible for infection, and are most important in the dissemination of the fungus in the soil.

Pathogenicity. Since the early work of Hartig, a great deal of attention has been given to the problem of the method of infection by this fungus. Early German workers concluded that the fungus would enter the uninjured roots of coniferous plants but that wounds were necessary for the penetration in the case of the broadleaf trees. While the rhizomorphs were considered important in the process of infection, it was not realized until recently that these organs alone could penetrate the root; that is, the mycelium is not capable of bringing about infection even on wounded surfaces. The work of Thomas, in America, and Van Vloten, in the Netherlands, has cleared up many points regarding the pathogenicity of the fungus and host resistance. According to Thomas, the tip of the rhizomorph, when it comes in contact with the root, becomes attached to the surface "by the hardening of mucilaginous substance which envelops the rhizomorph close behind its white tip." From the parent rhizomorph, side branches develop which penetrate the root, apparently by employing a combination of chemical and mechanical means. This penetration is not brought about by the growth of a few hyphae but by mass of the rhizomorphic branch acting as a unit. The suberized cells of the cork seemed to be partially dissolved by some enzyme, although its presence has not been definitely established.

Death of the cells takes place in advance of the penetrating rhizomorph, indicating the production of a toxic substance by the fungus. The rhizomorphs grow rapidly throughout the bark and kill large areas or completely girdle the root for some distance. The fungus also enters the wood following the medullary rays and often forms black lines.

Resistance to *Armillaria* root rot is known to occur in a number of varieties of fruit trees. This seems to be due, in some cases, to a mechanical walling off of the invading rhizomorph by the formation of a periderm layer below the point of infection. In other cases, this layer is in turn penetrated, but the fungus does not make a further extensive growth, as in the case of susceptible roots.

Van Vloten found decided differences in cultures of *Armillaria mellea* from different hosts. He emphasized the fact that certain strains rarely produce rhizomorphs, and since rhizomorphs are necessary for infection, these strains are not likely to be virulent when introduced into the soil. He found a difference in virulence in the various strains. Since he obtained different "strains" from the same host, he was of the opinion that one could not speak of an "oak strain" or a "coniferous strain."

The fungus grows well in culture and produces both rhizomorphs and sporophores, but as stated above, the production of rhizomorphs in culture depends upon the strain used.

Relation to Environment. *Armillaria* root rot is most likely to appear in orchards which have been planted in recently cleared land or near stands of timber. Often the old roots of forest trees remain for many years in the soil, and these may be responsible for local infections in the orchard. In California, it was observed by Hewitt that *Armillaria* infections are often localized along stream beds where old roots, stumps, etc., have washed down from above and lodged. Localized infected areas may remain so for a number of years, indicating that the fungus does not travel any great distance through soil free of roots. Heavy types of soil seem to restrict the growth of the fungus, but more important perhaps are the abundance of roots and the prevalence of decaying wood in any soil type.

Control. Infection on deep-seated roots and the fact that symptoms do not appear until the disease is well established render control of this disease very difficult. Some cultural methods may be employed for partial control. (1) When practical, a new orchard should not be planted on recently cleared land. (2) In an established orchard having a few diseased trees, it is worthwhile after removing the trees to dig a deep trench (6 ft) around this area. This trench should be kept open for several years. In so far as possible, all roots of the diseased trees should be dug up and burned. If healthy trees outside the trenched area show signs of dying, these should be promptly removed and the trenching extended.

Soil disinfection by the use of carbon disulfide or chloropicrin has proved successful. Carbon disulfide is preferable, as it is more effective and cheaper (see Chap. 1, page 23). This is injected into holes in the ground spaced about 18 in. apart and to a depth of 8 to 10 in., using 2 oz by weight (3/8 oz liquid measure) in each hole. The holes should be sealed to prevent escape. This treatment will result in the control of the fungus to a depth of 5 or 6 ft in normal soil. Any living trees in the treated area are killed, but since there is a limited lateral spread, injury does not occur much beyond the treated area.

Bliss, working on *Armillaria mellea* in citrus groves, expressed the opinion that carbon disulfide acted in an indirect manner. *Armillaria* is known to be parasitized by *Trichoderma viride.* Soil impregnated with carbon disulfide kills out many organisms competing with *Trichoderma* and allows this fungus to become established on the pseudosclerotia of *Armillaria,* which are not killed by the fumigant. This explains why high dosages of CS_2 are not as effective as moderate dosages, since the large doses kill out *Trichoderma* as well as its competing organisms.

Resistant root stocks on which some varieties of peach, plum, prune, and apricot can be grafted have been suggested. Among these are the plum stocks Myrobolan 29 and Mariana 2624.

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Powdery Mildew of Apple

While of minor importance in most commercial apple-growing regions, powdery or surface mildew is often a serious problem in nurseries and in certain regions where it is favored by climatic conditions.

History. Bessey first reported this disease from Iowa (1877), where he found it causing stunting of seedling nursery stock. It was reported from the eastern United States by Galloway in 1889 as a serious disease on young nursery trees. The fungus causing the disease was described and named by Ellis and Everhart in 1888. In 1914 Ballard and Volck gave a full description of the disease as it occurred in the Pajaro Valley in California, where climatic conditions were such as to cause extensive injury to apple orchards in this region. A study was made by Fisher of the mildew as it occurred in the arid regions of the Pacific northwest and contributed to the earlier work of Ballard and Volck on its control.

While the apple mildew fungus was known to occur in European countries, little attention was given to it, probably because of its minor importance.

Geographic Distribution. Apple powdery mildew appears to be widely distributed over the world wherever apples are grown. It has been reported from Japan, Australia, and New Zealand, as well as from most European countries. It is known to occur also in South America and Africa. The fungus has been found in practically all states in the United States and the provinces of southern Canada.

Economic Importance. Damage from mildew attack consists mainly in stunted growth. The foliage is dwarfed and twig growth reduced. In the nursery, seedling stock as well as young nursery trees are reduced in size and quality so that they are sold as "seconds." In the orchard, where the disease is severe, the shoots, blossoms, fruit pedicels, and the fruit may be severely damaged, but this is rare in most apple-growing sections.

Symptoms. Powdery mildew attacks 1-year-old shoots, affecting twigs, foliage, blossoms, and fruit.

Leaves. The disease usually makes its appearance in the spring as small grayish or white feltlike patches of mycelium on the underside of the leaves, which soon become crinkled and curled. Infected leaves take on a mottled appearance, which in some instances is evident before any fungous growth is visible. The fungus develops very rapidly, soon covering the entire leaf with mycelium and a powdery coating of spores. When very young leaves are infected they tend to increase in length but not in width, are stunted, and become folded longitudinally. Infected foliage is rendered hard and brittle and frequently is killed. In any case it ceases to function normally, and as a result the tree is devitalized directly in proportion to the extent and severity of infections.

Twigs. The fungus spreads rapidly from the leaves to the current season's growth, and in the early stages of development has the same general appearance on both. However, by midsummer the powdery condition of the twigs begins to disappear, and gradually the external grayish-white fungous growth is transformed into a brown feltlike covering in which numerous black fruiting bodies are embedded. Diseased twigs are either stunted or killed back, sometimes completely, sometimes only at the tips, which are especially susceptible to attack. Infected twigs that are not killed form a serious phase of the disease. New leaves on them are infected as rapidly as they appear. Likewise, buds formed on such twigs are infected, and in the spring the fungus resumes activity when the leaves and flowers unfold. The internodes of infected twigs are shortened, and the lateral buds, which tend to be elongated and purplish red in color, are bunched together. According to Baudys, severe infections of some varieties result in the production of witches'-broom.

Blossoms are usually infected from the overwintering mycelium in dormant buds, and as a rule the entire cluster with attendant leaves soon becomes diseased. Floral parts are shriveled and blighted, so that no fruit is produced. Fortunately, infected blossoms and leaves do not liberate conidia early enough in the spring to make a general "blossom blight" stage of the disease a likelihood. The first spread of spores from infected blossoms occurs when they open, which is usually a week or 10 days after normal buds.

Fruit is often attacked shortly after the blossoming period, but such infections seldom persist after the apple skin hardens, as it usually does by midsummer. Infections resulting from the germination of conidia are usually localized at the calyx end, whereas stem-end infections generally originate from the migration of the fungus down the pedicels. As a rule, infections of very young apples cause a stunting of their growth, along with the typical russeting which results from later fruit infection.

The russeting is characterized by a network of lines, sometimes so closely woven as to give the appearance of a solid patch. The infected areas harden, and as a result the growing fruit often cracks.

Causal Organism. Powdery mildew of apple is caused by *Podosphaera leucotricha* (E. & E.) Salm.[1] In eastern United States the closely related mildew *P. oxyacanthae* (DC.) De By. has been reported as the cause of a much less serious but similar disease.

When apple powdery mildew was first described by Bessey in 1877, he named the organism *Podosphaera kunzei*. In 1888 Ellis and Everhart described it as *Sphaerotheca leucotricha*. Burrill changed the name to *S. mali* (Duby), identifying the fungus with *Erysiphe mali* (Duby), which was later classified as *Phyllactinia corylea* (Pers.) Karst. The early confusion concerning the identity of the fungus was not cleared up until the work of Salmon was published in 1900. He definitely referred the fungus to *Podosphaera leucotricha.*

Hosts. In addition to apple, the most important host plant, *P. leucotricha,* attacks the pear and the quince. It has also been listed as a disease of cherry, plum, hawthorn, and serviceberry, but in the recent lists of diseases, *P. oxycanthae* is generally given as the cause of powdery mildew on these plants.

Disease Cycle. The powdery mildew fungus is an external parasite which spreads its filaments over the surface of the affected plant parts—leaves, flowers, young fruit, and 1-year-old stems. The pathogen is maintained by special sucking organs (*haustoria*), which enter the cell cavities of the underlying tissues. According to Csorba these haustoria penetrate directly through the epidermal cell walls. He states that there is a direct relation between the thickness of the outer walls and resistance, but that exceptions to this generally exist.

Summer spores, or *conidia*, are produced in great numbers from conidiophores, which arise from the prostrate vegetative mycelium. Each conidiophore may give rise to an unlimited number of spores which are ellipsoid, truncate, and hyaline and measure about 28 to 30 by 12 μ. Conidia which come in contact with host tissue germinate whenever environmental conditions are favorable. They are the chief means by which the fungus spreads once it has become established in the spring. Conidia germinate readily when the air is saturated and the temperature is between 50 and 77°F—the optimum being between 66 and 72°F. Leaves are susceptible to infection for only a short time after they emerge, but infections may occur at any time if the leaves are injured.

By midsummer the grayish-white fungous growth on infected areas starts to change to a brown color. Shortly after this change in color *perithecia* appear in the form of minute, dark brown, globular bodies, barely visible to the naked eye (75 to 96 μ in diameter). They are embedded in the external feltlike growth of the fungus. Each perithecium has 3 to 10 long rigid appendages on the upper side and short flexuous ones beneath. Each perithecium or fruiting body has one *ascus* which contains eight *ascospores* (22 to 26 by 12 to 14 μ). The hyaline, subglobose or oblong, single-celled ascospores reach maturity and are forcibly expelled during the warm spring rains. Perithecial formation on the apple seems confined largely to the twigs, but may occur also on petioles, midribs, and larger veins. On the pear they are formed mostly on the fruit.

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[1] Class Ascomycetes, Order Perisporiales, Family Erysiphaceae.

It is doubtful if the ascospores are of any importance in the overwintering of powdery mildew, although some workers believe they play a part. On the other hand there is little doubt of the importance of the dormant mycelium in the overwintering of the fungus. The importance of dormant mycelium and the unimportance of ascospores in the disease cycle of powdery mildew were pointed out as early as 1889 by Galloway and have been confirmed by many workers since. Because of the invasion of the buds by the fungus, mildew appears on the infected buds as soon as they open in the spring. From such initial infection points it spreads rapidly as a result of the dissemination of the conidia.

Control. The recommended methods of control of apple powdery mildew vary greatly in different regions. In nurseries the use of wettable sulfur sprays at proper intervals is sufficient to check extensive damage. In regions where spraying for scab control is not necessary, as in the arid sections of the northwest United States, microfine sulfur sprays in the prebloom and early postbloom sprays should be used. Sulfur sprays should be discontinued after high temperatures prevail.

Captan, glyodin, or ferbam will not give satisfactory control of powdery mildew. However, some organic fungicides are comparable to sulfur for powdery mildew control.

Varieties vary greatly in their resistance and the severity of attack. If possible, very susceptible varieties should be avoided where the disease is a factor in production.

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CHAPTER 4

DISEASES OF POME FRUITS (CONTINUED)

Sooty Blotch of Pome Fruits

Two superficial diseases of pome fruits, sooty blotch and flyspeck, are so often found together on the fruit that at one time they were considered to be caused by a single fungus. A more detailed study proved that two distinct species were involved. Sooty blotch is the generally accepted common name, but the terms "cloud," "sooty fungus," "sooty mold," and "sooty smudge" have been used.

History. It is probable that the fungus causing sooty blotch was present on some of its numerous wild hosts in North America and from these became established on cultivated fruits when they were introduced. Since it did not cause a serious decay of the fruit, it was rarely mentioned until buyers insisted on clean, showy fruit and discriminated against fruit marred by "cloud." We have an early account of the disease, however, in the writings of Schweinitz, who in 1832 gave a brief description of the fungus on apples in Pennsylvania. Two years later it was mentioned as occurring in France, but it is uncertain as to whether this report refers to sooty blotch or flyspeck or both. It was not until the end of the century that the disease, together with flyspeck, began to attract attention. Usually, these reports merely mentioned the disease as being common in certain years.

While either sooty blotch or flyspeck was reported in France in 1834, it was not until 1910 that Salmon reported it as occurring in England and expressed anxiety about this "new" disease imported from America. Again in 1916 he reported it for the first time on pears in England. This indicates that the disease might have originated in America.

Soon after the turn of the century sooty blotch was recognized as a serious disease in the cool, humid areas of eastern North America. In 1906 Clinton spoke of it as "one of the most serious fungous troubles of the apple in Connecticut."

Spraying for the control of the disease was inaugurated by Lampson in New Hampshire as early as 1894, and a number of control experiments were reported during the next twenty years mainly from eastern United States. During this period regular spraying for the control of apple scab and other diseases became a standard practice, and these fungicides in general also controlled sooty blotch.

In 1920 Colby in Illinois reported on a detailed study of a fungus causing sooty blotch, and after a careful study of its morphology and an analysis of previous descriptions, decided to erect a new genus which he called *Gloeodes*. Baines and Gardner in 1932 published an extensive report in which they established the host relations of the fungus and cleared up many points in regard to the time and manner of infection. In this same year Groves called attention to the various distinct types of the fungous colonies.

As stated above, interest in the control of the disease waned during the period when standard fungicides such as Bordeaux mixture and lime sulfur were applied along with lead arsenate in schedules which often extended into midsummer. Interest was renewed when the disease was found to be increasing to an alarming extent in some well-sprayed orchards where other diseases and insects had been adequately controlled. It was suggested that the change from the copper and sulfur sprays to organic fungicides was in part responsible for this upsurge of the disease. In addition, the control of insects by the use of DDT and insecticides other than lead arsenate may have proved a factor in lessened control since lead arsenate had been demonstrated to have mild fungicidal properties, especially when applied throughout the season.

Geographic Distribution. Sooty blotch on pomaceous fruits has been reported from North America, England, Continental Europe, Africa (Belgian Congo), and Australia. It is generally found in regions of high humidity and cool weather during the late spring and late summer or early fall months.

Symptoms. "Cloud" or "sooty smudge" are terms which adequately describe the general appearance of the disease on the fruit. Flyspeck often associated with sooty blotch consists of definite, circular black, often glistening spots resembling flyspecks in size and color, while sooty blotch spots are indefinite in outline, dull black to gray in color, and varying greatly in size (Fig. 32). The fungus is superficial, so that it can be removed by vigorous rubbing. A closer examination shows several types of colonies, some with rather loose feathery, or fernlike, strands, others with clumps or dots scattered over the thin sooty area, and others with thicker growth in which the strands are not plainly evident. There is no evidence of a definite lesion as in scab, but there may be a slight pitting beneath the fungus growth. It may be confused at times with the growth of fungi on honeydew deposited by aphids, but this sticky character of the honeydew is absent in the case of sooty blotch.

Economic Importance. The main loss from sooty blotch comes from the downgrading of the fruit. Buyers allow only a very limited amount of sooty blotch in No. 1 packed fruit. There is some evidence that fruit shrivels more rapidly when attacked by the sooty blotch fungus, but Colby was of the opinion that under identical storage conditions there was no perceptible difference between clean and blotched fruit in this respect. There seems to be no increase in the size of the blotched areas in storage.

Causal Organism. The fungus causing sooty blotch is now named *Gloeodes pomigena* (Schw.) Colby.[1] Because of its rather unique morphological characters and the confusion arising from its association with the flyspeck fungus, it has received a number of scientific names. It was first named *Dothidea pomigena* by Schweinitz in 1832. Other generic synonyms are *Asteroma* and *Phyllachora.* When the two diseases were considered as caused by one fungus, the name *Leptotherium pomi* was introduced, a name applied to what appears to have been the flyspeck fungus described by Montagne and Fries in 1834.

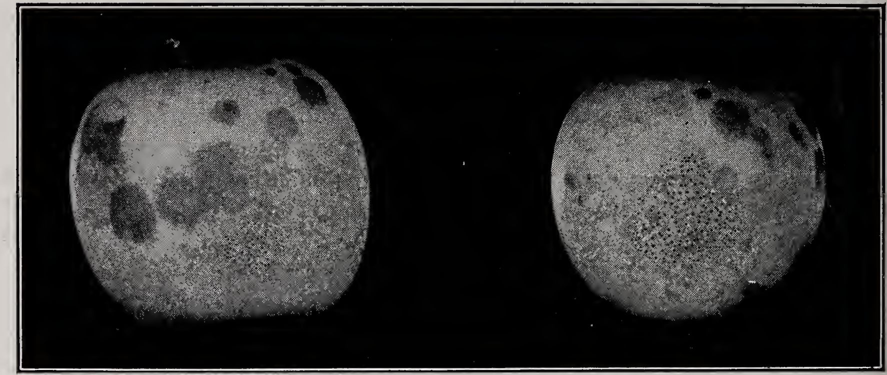

FIG. 32. Sooty blotch and flyspeck on apple fruit.

Hosts. *G. pomigena* is commonly found on a number of plants but is of economic importance only on the fruit of apples and pears. On wild hosts it has been reported on crab apple fruit and on the twigs of 22 other hosts, mainly trees and shrubs. Of these the most important in northern United States are sassafras, ash, *Smilax hispida,* elm, maple, sycamore, and hawthorn, since inoculations on apple from these hosts by Baines and Gardner were successful, and these plants commonly occur in the neighborhoods of the orchards. The fungus is also very abundant on wild blackberries, but inoculations on apple failed to produce the disease.

Morphology. The fungus forms a *thallus* of profusely branched hyphal threads. The individual cells are olivaceous and isodiametric in shape with slight constrictions at the septa. The short stubby lateral branches give the fernlike appearance to the thallus. The *pycnidia* are scattered throughout the thallus. They are flattened at the base (dimidiate), rounded above, and measure 20 to 40 μ in height and 70 to 100 μ in diameter. There is no distinct ostiole, the spores escaping by a rupture at the top of the pycnidium. While the pycnidia are abundantly formed, *conidia* are rarely found in them on the apple but are commonly produced on other hosts. The conidia are two-celled, hyaline, narrowly oblong to cylindrical, pointed at the ends, and measure 2 to 3 by 10 to 12 μ. They ooze out of the pycnidium by rupturing the membrane at the top of the pycnidium and collect in a gelatinous mass on the surface of the thallus. Chlamydospore-like cells are formed by the breaking up of the thick-walled hyphal fragments.

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[1] Class Fungi Imperfecti, Order Sphaeropsidales, Family Leptostromataceae.

The fungus is usually restricted to the outer surface of the cuticle, but Groves states that in some cases he has found the hyphae penetrating the cuticle and developing between the epidermal cell walls and the cuticle.

Disease Cycle. Baines and Gardner found that spores were produced much more abundantly on the young twigs of wild hosts than on the apple fruit. Thus it is probable that often initial infections are from this source. The fungus also develops on the twig of the apple, and as mentioned above, the old hyphal cells become thick-walled and break up. These chlamydospore-like cells may be washed to the developing fruit where they can start the formation of a thallus. Secondary colonies are started by a repetition of this process on the fruit after the initial spread of the fungus and enlargement of the infected area. New growth of the twigs of the hosts is infected during the course of the growing season, and thus the fungus overwinters.

Experiments by Baines and Gardner in Indiana and by Taylor and Bennett in West Virginia indicate that infections may occur at any time during the growing season but that cool, rainy weather, especially in May and June and in early fall, is essential for the development of the disease. It was found that temperatures around 65°F were most favorable for the growth of the fungus and that practically no growth occurred at 85°F. When May or June conditions are favorable for infection and temperatures are high during July and August, the disease may not appear on the fruit until 2 or 3 months later. Infections late in the season are likely to have a shorter incubation period if the humidity is high and temperatures comparatively low. Sooty blotch is often entirely absent or of no importance in years when hot, dry weather prevails up to near harvesttime. A combination of cool rainy spring weather with late summer rains and periods of low temperature in early fall is most conducive to severe outbreaks of this trouble.

Control. Sooty blotch is often found to be much more prevalent in low or shaded areas in an orchard than on the upper ridges and slopes where there is better air movement. Thus any practice which will open up the trees to facilitate quick drying will aid in control.

Fence rows and adjoining wood lots should be kept free of shrubs and undergrowth on which the fungus develops.

As stated above, very little difficulty was experienced with the control of sooty blotch during past years when the full standard inorganic pesticides were used for control of other diseases and insects.

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Flyspeck of Pome Fruits

While often associated with sooty blotch on the fruit, flyspeck is caused by a different fungus and may be found in the absence of sooty blotch. Flyspeck also occurs on a number of wild hosts along with or independently of sooty blotch.

The name flyspeck adequately describes the character of the individual spots. These slightly elevated black, definite, superficial spots usually occur in a group of from 6 or 8 to 50 or more (Fig. 32). Several such groups may be scattered over the surface of a single apple. While each dot seems independent of the others, a closer examination shows that a very fine thread connects them.

The specks on blackberry canes and shoots of wild plants often extend entirely around the stem and for a distance of several inches along the shoot.

It is of interest to note that there seems to be some antagonism between the two fungi, since it is rare to find sooty blotch encroaching on the area occupied by the flyspeck fungus.

Flyspeck is not as disfiguring as is sooty blotch and consequently is more likely to be overlooked in fruit grading if it occurs alone, but unfortunately this is rarely the case.

The fungus causing flyspeck for many years was called *Leptothyrium pomi* (Mont. and Fr.) Sacc. In 1953, Durbin et al. found the hitherto unrecognized imperfect stage *Zygophiala jamaicensis* originally described on banana. The perfect stage of this fungus is *Microthyriella rubi*. Petr.[1] This fungus, like sooty blotch, has an extensive host range in 15 families, including apple, carnation, banana, blackberry, and 17 wild hosts. Ascospore isolates from apples and wild hosts produced the perfect and imperfect stages when inoculated on young apple fruit. Conidial isolates from carnation produced flyspecks and the imperfect stage on apple. The disease cycle of flyspeck is much the same as that of sooty blotch, and it responds to climatic conditions in much the same manner.

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(See also references under Sooty Blotch.)

Leaf Blight and Fruit Spot of Pear and Quince

While generally known as "pear leaf blight," this disease has been called "leaf spot," "leaf scald" or "fruit spot," "quince leaf spot," "*Fabraea* leaf blight," and "*Entomosporium* leaf spot." The common name of leaf blight is not entirely satisfactory since it might be confused with the leaf blight phase of fire blight and furthermore does not indicate the commonly occurring fruit lesions.

History and Geographic Distribution. Leaf blight is known to occur in most countries of the world where pears and quinces are grown. It is probably of European or Asiatic origin since it has been known in Europe for well over a century. As the fungus is carried over winter on the twigs, it was probably introduced into North America from European shipments. It was recorded from both eastern and western United States as early as 1888 and was mentioned in horticultural writings much earlier. The fact that it was recorded only recently from Australia and New Zealand also indicates its probable European origin.

Symptoms. The disease is found on leaves, fruit, and shoots. On the *leaves* the spots appear first as small purple dots. These later extend to circular deep purple or dark brown lesions about $\frac{1}{4}$ in. in diameter. About the time the spots reach their maximum diameter, a small black pimple appears in the center of the spot. This is the spore-bearing body. When wet this takes on a glistening creamy appearance as a result of the gelatinous exuding spore mass. When the leaf spots are numerous, extensive defoliation results.

On the *fruit* the spots are black in color and become slightly sunken.

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[1] Class Ascomycetes, Order Hemisphaeriales, Family Microthyriaceae.

Otherwise they are similar to those on the leaves (Fig. 33). Cracking of the fruit is pronounced where the spots are grouped as is often the case.

The lesions on the *twigs* are observed on the current season's growth in midsummer or later. They consist of indefinitely outlined purple or black areas which by coalescing may form a canker several centimeters in length. Many of the superficial cankers are healed over, but some are found on the shoots in the spring of the year following infection.

Economic Importance. Susceptible varieties often are defoliated by midsummer, with dwarfing of fruit and reduction of fruit buds. Infected fruit is worthless since the fruit is disfigured and often cracked or misshapen. Twig injury in the orchard from the cankers is of minor importance since these cankers are not deep-seated and are healed over during the growing season.

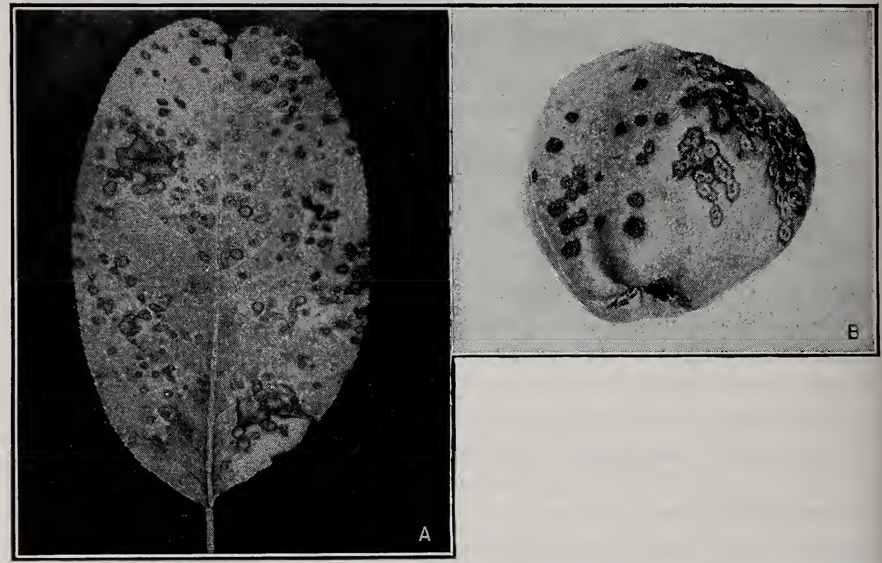

Fig. 33. *Fabraea* leaf blight on quince fruit (right), showing black, central fruiting bodies; at left, on pear leaf.

In the nursery, defoliation and twig infection are serious problems since they result in stunted growth of the nursery tree.

Causal Organism. The fungus responsible for the disease is *Fabraea maculata* (Lev) Atk.[1] The imperfect stage is *Entomosporium maculatum* Lev., the generic name being derived from the insectlike conidia. The perfect stage was originally described by Sorauer as *Stigmatea mespili*, but the generic name was changed to *Fabraea* by Atkinson, who was of the opinion that *S. mespili* was probably identical with *E. maculatum*.

[1] Class Ascomycetes, Order Pezizales, Family Mollisiaceae.

Hosts. While pear and quince are the hosts most commonly referred to, the fungus has been recorded on apple, Japanese quince, *Medlar*, *Sorbus* (mountain ash), *Amelanchier*, *Aronia*, *Cotoneaster*, and *Photinia*.

Disease Cycle. The perfect stage of the fungus develops on the fallen leaves and consists of a small disklike *apothecium*, leathery when dry but when wet becoming gelatinous. The *asci* when mature extend above the surface of the apothecium. The eight *ascospores* are hyaline and two-celled. In the *Entomosporium* stage the *acervuli* appear as black, subepidermal structures in the center of the spots on the leaves, fruit, or twigs. These break through the epidermis, exposing the mass of *conidia*, which are hyaline and measure 18 to 20 by 12 to 14 μ. The conidia are striking in appearance, since two of the four cells of which the conidium is composed are developed laterally at the connection between the two larger cells, and setae formed at their apex give an insectlike appearance to the conidia.

After the discovery of the perfect stage it was generally thought that the ascospores, formed on the fallen leaves, were responsible for the primary infection in the spring. Goldsworthy and Smith in 1938 reported on a series of experiments and observations which proved that most of the primary infection is from conidia developed on the cankers on the twigs. Attention had been called to this possibility by Sorauer as early as 1879, but no conclusive proof was offered. Only a few of the overwintering cankers produce acervuli the following spring. The acervuli which are formed beneath the epidermis of the host during February and March break through the epidermal layer, and the conidia are produced in April and May. These conidia are washed onto the developing leaves in the neighborhood of the active cankers. The primary infection is usually in a cone-shaped area below the active acervuli.

Plakidas reported in 1941 that under Louisiana conditions viable conidia in abundance were found on dead leaves in the spring and that the disease appeared later on the lower branches of Chinese sand pears. When the conidia-bearing dead leaves were suspended in the trees, infection occurred and no overwintering cankers were present on these trees. He failed to find ascospore development during the winter and spring. Thus it seems likely that some primary infection under certain conditions may be derived from the fallen leaves. The spots on the leaves become visible about a week after infection but enlarge rather slowly, so that the new crop of conidia is not produced until about a month later. The spread of the disease is very rapid when weather conditions are favorable, so that by early summer most of the leaves on susceptible varieties may be spotted. Dissemination of spores is thought to be by washing and wind-blown mist. It is noticeable that the early spotting and defoliation often appear in the top of the trees, and only later do the leaves in the lower part of the tree become infected. Also trees showing no primary infection but adjoining a heavily infected tree will show a spotting on the side next to this tree. Reinfections occur throughout the summer. The fruit may become infected as soon as it is exposed, but fruit infection is often entirely absent even where severe leaf infection is present. Twig infection may occur in the spring after the conidia appear on the developing leaves and may continue until late in the summer. The fungus remains alive in some of these cankers, and on these the new acervuli are produced in the spring.

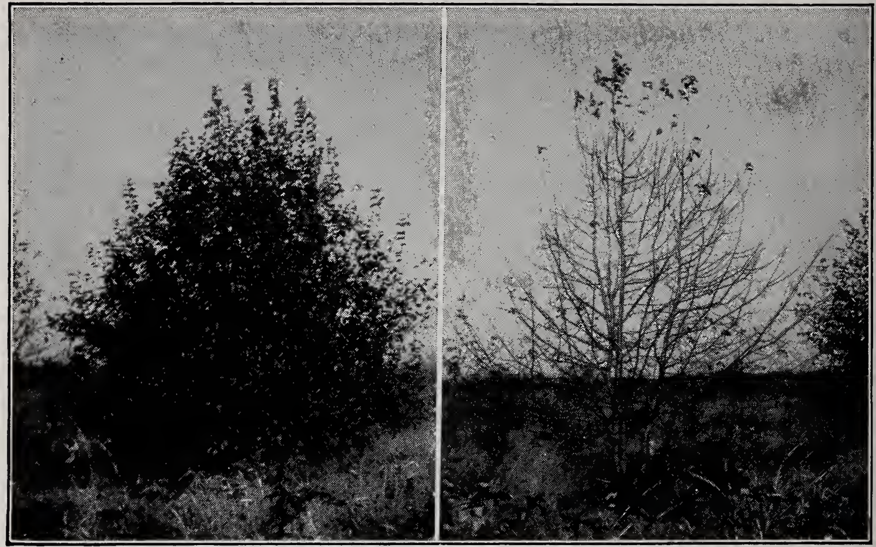

Fig. 34. Defoliation from leaf blight of pear. *Pyrus ussuriensis* seedlings, susceptible at right and resistant at left.

Varietal Susceptibility. It appears that most commercial varieties of pear and quince are susceptible to leaf blight. Clapp Favorite and Bosc have been recorded as more resistant than Seckel, Wilder Early, and Sheldon. Kieffer is moderately susceptible. In several years of observation on a group of seedlings of *Pyrus ussuriensis* at Urbana, Illinois, it was observed that these varied in susceptibility from some which were highly resistant, if not immune, to those which were so susceptible that they were completely defoliated by midsummer (Fig. 34).

Control. On the assumption that infected fallen leaves are the source of primary inoculum, sprays have been applied to the ground cover. These experiments were unsuccessful in controlling the disease, but did lead to a reinvestigation of the source of primary infection. Foliage sprays, if properly applied, will control the disease and prevent canker development. Successful results have been obtained in the past with lime sulfur and Bordeaux mixture, but more recently ferbam has been favored. Applications should be made early to prevent primary infection and continued on about the same schedule as for scab control on apples.

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Pear Scab

Pear scab resembles apple scab in many respects and is caused by a closely related fungus (*Venturia pirina* Aderh.). In fact, for many years the two diseases were supposed to be identical, but cross inoculations proved that each is confined to the one host. There are also certain morphological differences.

Pear scab is of much greater importance in European countries than in North America, in part owing to climatic differences and in part to the use of resistant varieties in America in regions favoring the disease. Pear scab is world-wide in its distribution but, like apple scab, is not prevalent in dry regions.

The symptoms and disease cycle are so similar to those of apple scab (page 26) that they need not be described, but certain differences may be mentioned. As stated under apple scab, twig infection on the apple is rare, while the pear scab fungus not only attacks the twigs but also causes serious damage to these organs. Thus the overwintering of the imperfect stage (*Fusicladium*) provides inoculum for the primary spring infection. In England the perfect stage in the fallen leaves is rarely reported and is probably of minor importance. On the European Continent, in North America, and Australia, the perfect stage is of considerable importance in initial infection. Leaf infection is not as extensive as in apple scab, but fruit and twig infections may be much more severe than apple scab under comparative environmental conditions.

Under the microscope the conidiophores are seen to be more zigzagged than those of *Venturia inaequalis.* The ascospores are similar to those of the apple scab fungus in being unequally two-celled, but the larger cell faces the apex of the ascus, while in *V. inaequalis* the opposite is true.

Most varieties of pear are susceptible to the disease but show differences in the degree of susceptibility. Unfortunately, there are distinct races of the fungus, consequently varieties recorded as resistant in one region may be susceptible to a different race in another region. In addition, the disease may be severe on the twigs of some varieties resistant to fruit infection. In the United States the Kieffer variety rarely scabs even in regions where apple scab is always present. In North America the varieties mentioned as especially susceptible are Flemish Beauty, Winter Nelis, Seckel, Anjou, Bosc, and Duchess, while Bartlett is less so. In Denmark Clapp's Favorite is listed as resistant, while in England it is considered susceptible.

Control. In most regions where apple scab occurs the same spray schedules may be used for the control of pear scab.

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Leaf Spot of Pear

Two leaf spotting diseases are commonly found on pears, one called "leaf blight," the other "*Mycosphaerella,* or *Septoria,* leaf spot." Leaf blight, considered the more serious of the two, is described on page 149.

History and Geographic Distribution. *Septoria* leaf spot was described in the United States in 1897. For many years it was regarded as one of the most important pear diseases, especially in the nursery. The perfect stage was demonstrated by Klebahn in 1908.

This leaf spot has been reported from practically all countries where pears are grown, but is usually absent in dry regions where the crop is grown under irrigation. Recent reports indicate that it has increased in importance in European countries where formerly it was regarded as a minor disease.

Economic Importance. The main damage results from defoliation. Often this occurs late in the season, so the devitalizing effect is not as serious as is the case with the leaf blight. Nursery trees may be defoliated before they have made full growth, thus reducing their market value.

Symptoms. This leaf spot is confined to the foliage. The mature spots are easily recognized by their grayish-white centers and well-defined margins. When these spots first appear, they are evident on the upper surface as small, brownish areas, lighter in color than the almost black spots caused by the leaf blight fungus. These spots enlarge somewhat, but at maturity are usually not more than ¼ to ⅛ in. in diameter. The margins are sharply defined. The center turns to a grayish color with a dark brown border (Fig. 35). On this gray center, small black scattered dots, the pycnidia, may be observed. In years unfavorable for infection only a few spots per leaf are found, but under some conditions the spots may be so numerous as to coalesce, in which case large leaf areas are blighted and defoliation results.

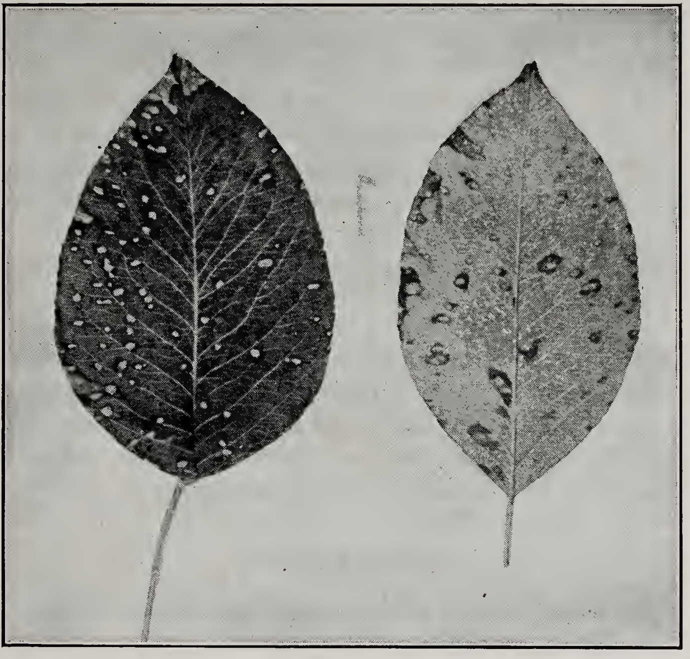

FIG. 35. *Septoria* leaf spot of pear; left, upper surface, right, lower surface.

Causal Organism. The fungus causing leaf blight is *Mycosphaerella sentina* (Fckl.) Schroet.[1] The imperfect stage of this Ascomycete is *Septoria pyricola*, under which name it was generally known until the perfect stage was found. The *pycnidia* are found on either surface of the leaf, but are usually more abundant on the undersurface. The *conidia* are filiform, hyaline, usually two-septate, and measure 40 to 60 by 3 μ. The conidia, in damp weather, ooze from the short ostiole, forming curled tendrils (cerri). The *perithecia* are formed on the fallen leaves and mature in the spring. They are in clusters, often without relation to the old lesions of the pycnidial stage. The black, short-necked perithecia are typical of the genus. The *asci* are clavate, 60 to 75 by 11 to 13 μ. The *ascospores* are hyaline, fusiform, one-septate, the two cells being about equal, measuring 26 to 33 by 3 to 4 μ.

Disease Cycle. The ascospores are discharged from the fallen leaves and are carried to the developing leaves. So far as known, primary infection results only from the ascospores. The spots develop rather slowly, and the pycnidia are not formed until about a month after infection. Secondary infection results from the washing of the conidia after they ooze from the pycnidia. Little is known about the method of penetration, but it is assumed to be mainly through the stomata. Infection from conidia may continue throughout the growing season, but only when abundant moisture is present to distribute the inoculum. The build-up of infection is often delayed until late-summer or fall rains occur. The life cycle is completed by the formation of the perfect stage on the fallen leaves during the fall and spring.

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[1] Class Ascomycetes, Order Sphaeriales, Family Mycosphaerellaceae.

Hosts. Pear and quince are the common cultivated hosts of this fungus. It has been reported on apple and several species of pear other than *Pyrus communis*. Differences in susceptibility of varieties of pear are marked. Seedlings in nursery rows are often severely attacked, while the cions in neighboring rows may show few leaf spots. Bosc, Anjou, Bartlett, and Seckel are standard varieties which are quite susceptible, while Flemish Beauty, Duchess, and Winter Nellis are more resistant, and Kieffer very resistant.

Control. Sprays applied for the control of *Fabraea* leaf blight or pear scab usually are effective in keeping down this leaf spot. In France eradicant sprays applied for leaf blight and scab control were effective in controlling *Mycosphaerella* leaf spot. Ordinarily this leaf spot is not serious enough to warrant special control measures.

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Blossom Blast of Pears

A blossom blight, twig blight, and canker of pears caused by *Pseudomonas syringae* Van Hall is likely to be confused with fire blight on this host.

This disease, or a similar one, has been reported from South Africa, England, and the United States (Arkansas and California).

The *symptoms* on pear resemble those of fire blight. Dormant buds may be killed, or the blossom cluster may wither as it expands in the spring. In this case, the fruit spur will be found to have been invaded, probably through the fruit-stem scars during the autumn or early winter months. Superficial cankers are produced on the young shoots. The growth of the periderm the following year causes cracking of the outer bark of these cankers, giving them a roughened appearance.

The disease may cause a spotting of the leaves and blisters or pits on the fruit, similar to the blister spot of apple, but this rarely occurs.

Wormald in England found that three types of bacteria could be obtained from the pear-blasted blossoms on twigs. He agreed with Wilson that it was possible that these were variations in the organism responsible for bacterial canker or gummosis of stone fruits. The nomenclature of this organism is discussed in some detail under Bacterial Canker and Gummosis of stone fruits. (See also Blister Spot of apples.)

It appears from the work of Wilson and of Wormald that infection by this organism occurs during the winter and early spring months.

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Nonparasitic Diseases of Pome Fruits

The large number of fruit diseases of nonparasitic origin is not surprising in view of the tremendous variations of habitat and cultural practices to which most fruits are subjected. They are produced on soils which vary greatly in acidity, type, fertility, and moisture content. Likewise they are subject to extreme fluctuations in rainfall, temperature, humidity, sunshine, and air movement, all of which have a direct bearing on the normal physiological processes of the crops involved. Still other problems arise as a result of irrigation, spraying, pruning, and other such practices. While the variables mentioned do not comprise a complete list, they are sufficient to emphasize that one or several may operate to the detriment of normal plant growth and, accordingly, may actually produce a diseased condition. Only those nonparasitic diseases of wide distribution, with well-marked symptoms, will be discussed in detail. A list of minor troubles due to weather conditions, mechanical injury, genetic factors, etc., will be listed with a brief statement of symptoms in order that they may be contrasted with those of the more important nonparasitic troubles.

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Bitter Pit

The disease known rather generally as bitter pit is also called "Baldwin spot," "fruit spot," "apple brown spot," "smallpox," and "fruit pit."

German workers know the disease as "Stippen," "Stippflecke," and "Stippigwerden." The French know it as "liège," or "points bruns de la chair."

The occurrence of a disease similar to bitter pit was reported in Germany in 1869, although it was known by growers long before this date. It was next reported from Australia in 1886 and from the United States in 1891. The disease seems to be present wherever apples are grown, but causes greatest losses in Australia and the United States.

Economic Importance. The losses from bitter pit occur in the orchard, in transit, and in storage. Because of the inconspicuous indications of the "pits" on the exterior, many diseased apples are packed, and it is only after a period of storage that the symptoms become so marked that the fruit is considered off-grade or worthless. It is not an easy matter to estimate the damage done by this disease, especially since in many cases the growers do not recognize it as a specific disease but ascribe the damage to hail or other factors. On a susceptible variety, as high as 85 per cent of the fruit may show the disease under favorable conditions for its development. It is only under such conditions, however, that bitter pit is of any great economic importance. The most extensive losses occur in those regions where the apple is grown in an environment unsuitable for apple production, such as under irrigation, where wide fluctuations in rainfall occur during the growing season or where unusual soil types influence water intake. It is most serious on young trees and on older trees producing a light crop. Under conditions favorable for tree growth and fruit production, the loss is negligible.

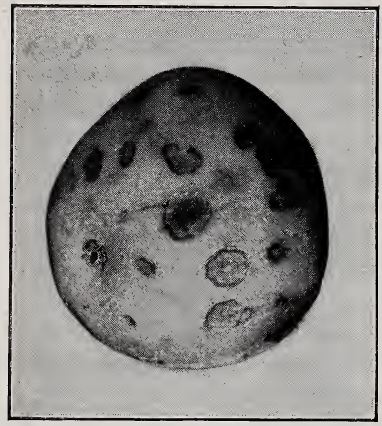

Fig. 36. Bitter pit of apple, advanced stage.

Symptoms. Bitter pit occurs only on the fruit. This disease, in contrast to "cork," does not appear until the apples are over half grown and often not until they approach maturity. The first evidence of the disease is the presence of slightly sunken, water-soaked, circular spots. These sunken areas differ in color from the surrounding tissue. On a yellow or green surface they are dark green, while on a red surface they are a duller, deeper red than the "blush" of the fruit. There is no external browning of the skin in the early stages of the disease, and frequently this does not occur until after the fruit is in storage (Fig. 36). The number of the "pits" varies from two or three to a hundred or more. They are not centered about the lenticels as is the case in Jonathan spot. Usually they are more numerous around the calyx end and frequently appear on only one side of the fruit but may be rather evenly distributed.

Internal Symptoms. Beneath the depressions on the skin are to be found spongy masses of dead tissue, light to dark brown in color and hemispherical in shape. Unless invaded by rot-producing fungi the tissue is dry and spongy. The depth of the dead area varies from a few cells to ¼ in. or more, but is always deeper than in Jonathan spot. Necrotic spots are often, but not always, found in the deeper flesh of bitter pitted apples. These, as well as the border pits, have a rather definite relation to the vascular bundles, as will be pointed out in the discussion of the cause of bitter pit. It is claimed by some that the internal brown spots are produced previous to the formation of the superficial areas. The deep-seated brown areas are likely to be confused with "cork spot."

In storage the skin above the spongy superficial spots dies and the areas become brown and more decidedly sunken. Often *Alternaria* and other fungi invade the tissue, and true rots are produced.

Cause. Numerous theories have been advanced to explain this disease. It has been fully established that it is not due to parasitic organisms. That it is not due to deficiency of the common plant nutrients has been proved by extensive fertilizer experiments in Australia and elsewhere. The generally accepted explanation at the present time is that the disease is due to a *disturbed water relation.* Two other causes have been advanced within recent years: (1) virus and (2) boron deficiency.

Regardless of the *primary cause* of the disease the manifestations of the symptoms seem to be due to an abnormal water relation in the cells of the fruit in the neighborhood of the vascular strands which permeate the apple flesh. This vascular system, which enters through the stem, is the sap-conducting system which carries the water and nutrients to all parts of the apple fruit. After entering the apple from the stem, the main strands grow up around the core. From these arise numerous smaller branches which form a network throughout the pulp of the fruit. Many of these smaller branches extend to the flesh just beneath the skin. The function of this elaborate system is to furnish water and nutrients to the pulp cells which compose the main part of the flesh of the apple. These pulp cells are large, thin-walled sacs, stored with elaborated food, which in a soluble form is carried in part to and from these cells by the vessels of the vascular system. From 75 to 90 per cent of the mature apple is composed of water. Loss of water from the apple is regulated by the stomata (breathing pores) and lenticels which break the continuity of the waterproof skin of the fruit. Thus the apple fruit may be regarded as a complex living system which may be "thrown out of gear" by any unusual environmental condition.

A microscopic examination of the diseased areas of bitter pit shows the presence of a number of collapsed dead cells which are rather closely associated with the vascular bundles, especially those which end in the periphery of the apple. The internal necrotic areas are generally closely associated with the larger vascular bundles. The theory that bitter pit is due to a disturbed water relation is supported by the fact that the collapse of these cells appears to be correlated with unusual weather conditions such as dry hot weather followed by a period of excessive moisture. In irrigated regions, light irrigation during the early season followed by heavy late irrigation is supposed to favor the disease. Oversized fruit, especially on young trees, seems to be unusually susceptible to the disease. Heavy pruning also seems to increase the predisposition of the fruit to bitter pit. All of these factors indicate that the disease is the result of an unbalanced condition in the development of the fruit. Several theories have been advanced to account for the actual collapse of the cells. One theory is that when abundant water is supplied following a dry period there is a rapid intake of water into the fruit, the pulp cells are unduly distended, and a certain number burst. Another is that under the same conditions the excessive pressure of some of the distended cells actually crushes the neighboring cells. Still another theory is that, owing to differences in osmotic pressure, water is withdrawn from some of those of low osmotic pressure by surrounding cells and there is a collapse of the former.

The virus origin of the disease has been recently supported by Atanasoff, who has found typical mosaic patterns on the leaves of trees on which bitter pit occurs on the fruit. He maintains that the nonparasitic diseases known as "blister drouth spot," "cork," "crinkle," and the more generally restricted "bitter pit" are all different manifestations of one disease. Atanasoff did not prove by well-controlled experiments the important point as to transmission of the disease to healthy plants by grafting. The leaf mottling described by Atanasoff is evident only during the early period of leaf development and disappears later in the season. It consists of an indistinct "clouding" to a more or less "ring spot" type of mosaic in which the affected areas are lighter green in color than the surrounding tissue.

The theory that boron deficiency is the cause of bitter pit was first suggested by Atkinson and Askew as a result of experiments in New Zealand on the "internal cork" of apples. Various other investigators in Canada, United States, England, and Australia have reported favorable results from the addition of boron to the soil and to the trees. The reports indicate that the disorder known as "internal cork" can be controlled by the use of boron, but in recent experiments in England, it was found that bitter pit as here defined did not yield to boron treatment. The positive results secured by the use of boron on internal cork, together with the tendency of a number of investigators to regard all of these flesh necrotic lesions as being due to the same factor, is responsible for the extension of the boron-deficiency theory to cover bitter pit.

Control. Since bitter pit is supposed to be directly or indirectly caused by irregularities in the moisture or nutritional requirements, one suggested method of control is to modify the cultural practices so as to assure an even supply of water and nutrients to the tree during the growing season in so far as this is practical. Some of the orchard-management suggestions which have been made are: (1) Follow cultural practices which will ensure an even distribution of soil moisture. (2) Avoid excessive pruning in any one season. (3) Avoid excessive use of nitrogenous fertilizers in an otherwise productive orchard. (4) In thinning remove fruit from weak spurs and avoid excessive thinning since oversized fruit is more subject to the disease. (5) In irrigated sections avoid overirrigation in the latter part of the season and attempt to regulate the water supply so as to give an even growth of the fruit throughout the season. In planting a new orchard in regions where bitter pit is known to be a problem, those varieties should be avoided which are known to be especially susceptible to the disease. While a number of lists of susceptible and resistant varieties have been compiled, these are of little value to the commercial grower. A variety which is regarded as quite susceptible in one region may be relatively free of the disease in another. Baldwin, Cleopatra (Ortley), and Stayman have been generally regarded as susceptible wherever these varieties are grown.

Early picking is regarded as conducive to the development of the disease in storage, and consequently this practice should be avoided, especially in a year when there have been heavy later rains and some bitter pit is known to be present in the orchard. Often bitter pit is inconspicuous when the fruit is harvested but becomes more apparent after the fruit has been in storage for a time. It has been found advisable to keep the fruit at an even low temperature in order to reduce the loss in storage. The condition is much worse in common than in commercial cold storage. *Alternaria* and other rot-producing organisms enter the pitted areas under conditions favorable for the development of those fungi. This loss can be reduced by early sale and use of the pitted fruit.

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Jonathan Spot

Jonathan spot is primarily a skin disease closely associated with the lenticels of the fruit. It frequently occurs in the orchard on mature apples but is more commonly observed on picked fruit. It is found throughout the world where this variety is grown. Occasionally it occurs on other varieties such as Rome Beauty, Wealthy, and King David, but is predominately a Jonathan disease.

The loss from Jonathan spot is enormous in seasons when it is prevalent and especially when the fruit is held in common storage for some days after picking. Not only does the spot mar the fruit, but also *Alternaria* and other rot-producing fungi gain entrance in the spots. Records compiled by the U.S. Bureau of Markets show that for this variety 13.1 per cent of the boxed apple crop and 16 per cent of barreled fruit have Jonathan spot.

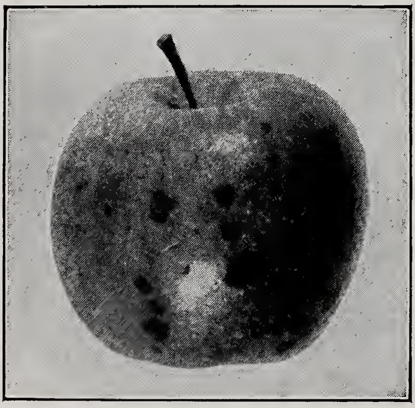
FIG. 37. Jonathan spot.

The spots are typically from 1/16 to 1/8 in. in diameter, deep brown to black in color, slightly depressed, circular in outline, and with a definite, clear-cut border (Fig. 37). They are more abundant around the stem end of the fruit and are usually associated with the lenticels. In storage the spots increase in size and often become more irregular in outline. Unlike bitter pit, the epidermal cells are dead and the corky area beneath extends only a few cells into the flesh. Jonathan spot is more prevalent on highly colored than on undercolored apples, and on the individual fruit are more numerous on the blush side.

Jonathan spot is undoubtedly a physiological disease associated with the metabolism of the apple as it approaches maturity. The fact that the spots are associated with deep color indicates some relation to the metabolic changes which occur in this region. Reducing the acidity of the cells containing the colored pigment by means of ammonia fumes changes the color from red to a deep purple and gives support to the theory that the reduction of the acid content of the superficial color-bearing cells is responsible for the symptoms of the disease.

Jonathan spot does not yield to oil-impregnated-paper methods employed in the control of scald. Evidently it is not due to the same cause. The recommended methods of control consist of harvesting the fruit as soon as it reaches proper maturity and prompt removal to cold storage. A delay of 2 to 3 weeks may result in 25 to 50 per cent of the fruit becoming spotty as compared to 2 to 3 per cent in fruit harvested at the proper time and removed immediately to cold storage.

Jonathan freckle is regarded as distinct from Jonathan spot by Heald, who described it as consisting of "circular areas of discolored tissue up to ¼ inch in diameter, only skin deep and not becoming depressed. Appears only in storage."

He states that it shows no relation to the lenticels as is the case with Jonathan spot. [F. D. Heald, Nonparasitic diseases of the apple in Washington, *Proc. Wash. State Hort. Assoc.*, **16**:6–7 (1920).]

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Internal Cork

This disease is often confused with bitter pit, and some investigators believe that the two are due to the same physiological conditions. Recent investigations, however, indicate that cork and certain related diseases may have a different origin from that of bitter pit. The name "cork" as here used includes also such diseases as "corky pit," "corky core," "spot necrosis," "drouth spot" (of Mix), and "crinkle." "Die back" and "rosette" of the twigs as defined by Mix are also included, although these terms have been used to describe physiological troubles of different causal origin.

Cork has a world-wide distribution, but like bitter pit, it is of commercial importance in only a few localities. These localities do not always coincide with those where bitter pit is prevalent, and this fact has been used as an argument in favor of different causes for the diseases.

The following quotation from Burrell summarizes the symptoms as observed in Lake Champlain Valley in New York:

> *Drouth spot* is a fruit symptom characterized by large, more or less superficial necrotic areas which ultimately become russeted, retarded in growth and cracked. It usually appears before the fruit is half grown. Drouth spot is sometimes confused with spray injury.
>
> *Internal cork* is characterized by masses of dead cells at any point in the flesh of the fruit. If it appears within about six weeks of petal fall, the lesions become dry, corky and definitely delimited. If it is early and severe, the fruits may be badly distorted. If it appears in midsummer or later, the lesions are spongy rather than corky, their margins are less sharply defined and the affected fruits are normal or nearly so in shape.

Die back and rosette refer to conditions of the terminal twigs. Die back, in an early stage "incipient die back," or scorch, is characterized by a reddening and scorching of the leaves and, later, the death of the terminal portion of the twig.

> When fruit symptoms appear within 6 weeks after petal fall, the symptom is fairly constant for any given variety. However, there are wide differences between varieties. Fruits on Tolman Sweet alone, among the sorts generally grown in the region, are consistently free from both drouth spot and internal cork, although Tolman Sweet trees, in rare instances, show rosette and die back. The usual symptoms on fruits of McIntosh, Northwestern Greening, Rome Beauty, Northern Spy, Winter Banana, Cortland and Lobo is drouth spot. The usual symptom in Fameuse, R. I. Greening, Duchess and Wealthy is internal cork. Northern Spy and Duchess fruits often have both cork and drouth spot or a combination of the two.

When the disease appears early in the season, many of the fruits drop. Those which remain on the tree sometimes become decidedly distorted and wrinkled, especially at the calyx end. This condition is called "crinkle." Carne and Martin report that crinkle is always preceded by a glassiness of the tissue which resembles "water core."

The *cause* of internal cork and the related diseases was at one time thought to be due to the same conditions which brought about bitter pit, that is, a disturbed water relation. Atanasoff believes that the two diseases are identical and that a virus is responsible. Within the last ten years definite proof has been obtained that internal cork can be prevented by the addition of boron to the soil, by injection into the tree, or by spraying the leaves with a boric acid solution. Other chemicals have failed to give this response. The beneficial effect of boron in such widely separated regions as western Canada, New York, West Virginia, Nova Scotia, Finland, Australia, and New Zealand strongly indicates that internal cork and related diseases such as drouth spot, crinkle, rosette, and die back are due to boron deficiency.

Various methods have been employed in applying the boron. In New Zealand, Askew and Chittenden used three methods of application: (1) soil treatment with 50 to 100 lb of hydrated borax to the acre, (2) injection with a 0.25 per cent hydrated borax solution, and (3) spraying the trees with a 0.1 to 1 per cent solution. McLarty in British Columbia used the dry salt of boric acid in auger holes in the trunks and secured good results, but with some local injury. He used approximately 1.5 g per 100 sq cm of the cross-sectional area of the tree. In Finland, Jamalainen secured good results by the use of 200 to 400 g of boric acid applied to the soil immediately following bloom.

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Water Core

Water core occurs both in the orchard and in storage. It is a condition which is especially influenced by environmental conditions during harvest and storage. While it is usually understood to be prevalent only during the maturing phase of the fruit development, it is known to occur at very early stages, especially in relation to the disease known as "crinkle." The relation of this early type of water core to the late phase has not been adequately investigated and will not be treated in this discussion (see Internal Cork). The name "water core" is derived from the fact that the tissues involved have a clear, glassy appearance and the location is usually, but not always, around the core of the apple. The disease has been observed in apples from all parts of the world.

The loss from water core is not primarily due to the conditions of the tissues, since the cells involved have the normal taste and firmness of the remainder of the apple. Under proper storage conditions the affected apples may return to the normal condition. It has been proved, however, that apples having water core are much more subject to internal breakdown, browning, and soft scald than those free from this disease.

Symptoms. Water core is more likely to be found on fruit from exposed portions of the tree where it has been fully exposed to the sun and where temperatures are high. It is not easy to detect water-cored apples, but frequently they may be distinguished by an unnatural blush or dull area on the exposed face of the fruit. When such fruit is cut open, certain areas in the flesh, usually around the vascular strands, are "glassy" in appearance. The tissue is not soft, but on the contrary may be firmer than the surrounding flesh. The affected areas may appear in a regular arrangement about the core or, more frequently, only in the exposed half. Their size and position vary from quite small definite spots to large irregular areas which may extend from the core almost to the skin. Radial lines of water core tissue may extend through the flesh, following the main vascular strands from the core to the periphery of the fruit. Water core is most evident after the fruit has been harvested and kept at relatively high temperatures for some days.

Cause. At one time it was thought that excessive water supply near the end of the growing season was responsible for water core in that the tissues of the fruit became gorged with water near the vascular system. This theory has been disproved by irrigation experiments where it was determined that lightly irrigated trees developed more water core than those heavily irrigated. An excess of soil moisture late in the season did not increase the amount of water core. It has been proved that excess sap concentration is responsible for the development of water core and that this occurs more frequently on apples exposed to high temperatures. Late-picked apples show a decided tendency to develop water core since sap concentration increases with the maturity of the apples. The disease is apparently due to sap exuding from the cells into the intercellular spaces under pressure.

Control. Water core can be controlled to a certain extent by cultural practices, and when it develops, its injurious effect can be reduced by proper handling of the fruit. The production of good foliage which will shade a high percentage of the fruit and thus reduce excessive exposure to high temperatures will reduce water core to a negligible quantity.

Where conditions are such as to favor the development of water core, the fruit should be picked at an early stage of maturity and should be placed under cool or cold conditions as soon as possible. Under such conditions the disease disappears from the affected fruit but in some cases is followed by an internal browning if held too long in storage.

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Scald

The general term "scald" is applied to any indefinite superficial browning of the apple. "Soft scald," "deep scald," and "soggy breakdown" are terms used to indicate types somewhat different from "common scald."

Scald is exclusively a storage disease and usually develops several weeks after the fruit is harvested. It has been reported from all regions where the apple is grown or marketed. Undoubtedly scald has been known to fruitgrowers since apples were stored, but it did not appear to be distinguished as a special trouble until fruit was transported long distances and held in storage over long periods. As early as 1896 it was regarded as one of the most serious disorders of the apple in the United States. The shipment of apples from northwest to eastern United States markets call attention to the enormous losses which might occur from this trouble since high-grade, well-packed fruit, when placed on the market, was often so badly affected as to be an almost total loss. A series of experiments on the cause and control of scald was started about 1916 by workers of the U.S. Department of Agriculture, and by 1919 it was established that the disease was directly due to the production of volatile esters. When these were absorbed by oil, the injury was reduced to a negligible quantity.

Economic Importance. In the past, losses from scald have been enormous because of the fact that the disease did not develop until the fruit was actually placed on the market, when within a few days the entire lot would be rendered practically worthless. Thus the expense of harvesting, packing, transportation, and storage would be involved in the loss. Since the marketing agencies did not always understand the cause of the trouble, many lawsuits were started in which the storage agents were made the defendants on the grounds that the fruit had been "frozen" by the careless operation of the storage plant. Since the use of oil wrappers has come into general use, the losses have been greatly reduced, especially in fruit which goes through the regular marketing channels. In addition to direct loss from scald, there is often a serious loss from rot fungi which become established in the scald-injured fruit.

Symptoms. The first appearance of scald consists of a slight deepening of the color of the skin which may not be at all evident in darker-colored varieties. It is usually more pronounced on the lighter-colored areas of the apple and on undercolored fruit. Later the involved areas turn to a light brown. The border between the affected and normal tissue is not sharply defined except in the case of certain types of "deep" scald. At first the brown discoloration is confined to the surface of the skin and there is no evidence of softening. Later the deeper layers of the skin become involved and softening becomes apparent. At this stage the skin sloughs easily, so that handling of the fruit exposes the flesh to rot-producing fungi. The surface becomes darker as the scald ages. Usually scalded areas are quite large, often involving from half to three-fourths of the entire surface.

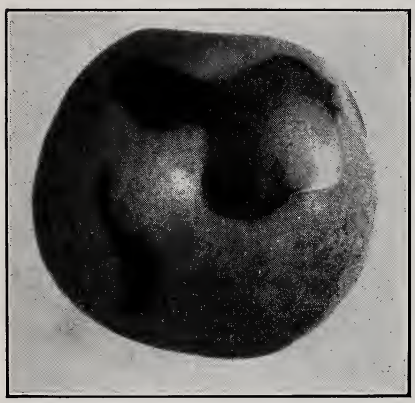

FIG. 38. Soft or deep scald of apple, showing regular outline.

In the case of deep scald, the outline of the spots is very definite and the pulp cells are involved for some distance into the flesh. The spots of deep scald soon become sunken with the collapse of the pulp cells, which are very soft and watery (Fig. 38).

Varietal differences in relation to common scald, soft scald, and soggy breakdown are marked. Differences in conditions under which the fruit is harvested often give an erroneous impression of the relative susceptibility of varieties to these diseases. Plagge, Maney, and Pickett record varietal susceptibility as follows:

Common Scald. SUSCEPTIBLE: Arkansas, Winesap, Stayman, Grimes, Ben Davis, York, Rome, Rhode Island Greening, Wagener, and Baldwin.

RESISTANT: Jonathan, Wealthy, Ralls, Willow, McIntosh, Golden Delicious, Northwestern Greening, King David, and Fameuse.

Soft Scald. SUSCEPTIBLE: Jonathan, Northwestern Greening, Rome, Wealthy, and Golden Delicious.

Soggy Breakdown. SUSCEPTIBLE: Grimes Golden, Wealthy, Golden Delicious, Northwestern Greening, Jonathan, Rome Beauty, and Winter Banana.

RESISTANT: Winesap, Stayman, Delicious, Willow Twig, York Imperial, Baldwin, Rhode Island Greening, McIntosh, Ben Davis, Fameuse, and Arkansas.

Cause. Scald has always been considered a physiological disease, and the manner in which it developed following storage suggested to the early investigators that it was due to some condition which existed in the storage. At first it was suspected that it was due to an excess of carbon dioxide which always occurs in cold-storage plants. Brooks and his coworkers in the U.S. Department of Agriculture disproved this theory by subjecting apples to amounts of CO_2 far in excess of what occurred in storage under conditions where the air was circulated. Other common gases were tried with like results. The free circulation of air in the cold storage prevented the development of scald. All these facts pointed to the probable cause being the accumulation of certain volatile materials produced by the apple. It was known that certain esters were given off by the apple, and it was found that scald could be artificially produced by exposing the fruit to vapors of ethyl or amyl acetate or methyl butyrate. When the air of the storage is kept in circulation or changed at frequent intervals, these esters are not allowed to accumulate and scald does not develop.

Control. Before the cause of apple scald was known, the practice of protecting the fruit from scald by air circulation in storage was common. After the discovery of the real cause it was evident that the absorption of the volatile esters by some material which would not injure the fruit would offer the best solution for control. As early as 1903 it was found that paraffined wrappers greatly reduced scald over common paper wrappers. Oil-impregnated wrappers were tried and were found to be very successful. Shredded oil paper placed loosely around the fruit was also found effective. At present most apples which are not wrapped are packed with the shredded oil paper, and as a consequence the losses from scald have been greatly reduced.

The storage temperature most favorable for the development of scald is around 60°F. Where air circulation is poor, apples held at or near this temperature may develop scald even with oil wrappers. For this reason the fruit should be held at much lower temperatures. Fruit should be picked at the proper stage of maturity, since that harvested before reaching maturity is more likely to scald.

Soft scald and soggy breakdown of apples are more difficult to control by oil than common scald. These diseases are definitely influenced by prestorage conditions and the temperature at which the fruit is held in storage. Delayed storage greatly increases both diseases. Fruit held at 32°F tends to develop more soft scald than when held at 35°. Coating the apples with a 50:50 oil-paraffin mixture or a treatment with 20 to 30 per cent carbon dioxide for a few hours before storage gives satisfactory control of both soft scald and soggy breakdown.

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Nonparasitic Diseases of Minor Importance

Mealy breakdown is characterized by mealiness, a senile condition which may be postponed by proper handling and storage. Attempts to hold fruit beyond the normal storage period often result in mealiness.

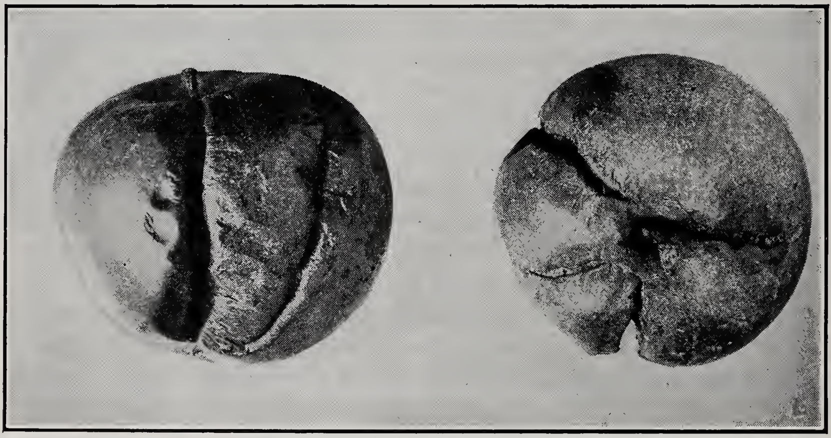

Fig. 39. Weather injury, cracking of Stayman Winesap following rainy weather.

Brown heart is supposed to be due to the accumulation of carbon dioxide. [F. Kidd and C. West, Brown heart—a functional disease of apples and pears. *Dept. Sci. Indus. Research, Food Invest. Bd.* (*London*) *Special Rept.,* **12**:1–34 (1923).]

Freezing injury, with effects similar to those of scald, may result from handling fruit while the tissues are frozen.

Shriveling results from storage under low humidity conditions or when certain varieties are held beyond the normal storage period. This condition can be delayed by oil treatment when fruit is stored. Apples russeted by improper spraying or weather conditions are especially subject to shriveling.

Internal browning of Yellow Newtown apples is characterized by brown streaks in the pulp radiating out from the core. It appears to be a cold-storage disease closely related to scald. [E. L. Overholser et al., Factors influencing the development of internal browning in Yellow Newtown apple. *Calif. Agr. Expt. Sta. Bull.*, **370**:1–40 (1923).]

Leaf scorch is characterized by marginal burning or scorching of leaves. This condition appears to be associated with a lack of potash, although other factors sometimes contribute. [T. Wallace, Leaf scorch on fruit trees. *J. Pomol. Hort. Sci.*, **7**:1–31 (1938).]

Physiological cracking is a nonparasitic disease of apple which is usually brought about by excessive rains following dry periods. It is especially serious on some varieties, such as Stayman Winesap, where as much as 50 per cent of the fruit may be rendered worthless (Fig. 39).

Sun scald of apple and other fruits occurs where fruit is exposed to the direct rays of the sun during excessively hot weather. Defoliation as a result of leaf fungi or spraying often exposes fruit which is not sufficiently hardened to the sun's rays, and excessive browning of the skin results. Often a corky layer of cells is formed beneath the exposed surface.

CHAPTER 5

DISEASES OF POME FRUITS (CONTINUED)

VIRUS DISEASES OF POME FRUITS

The virus diseases of pome fruits are here treated as "minor diseases" partly because of their economic unimportance and partly because they have not been extensively investigated. The potential importance of these diseases, however, should be recognized.

Apple Mosaic

Variegation of apple foliage has been recorded by a number of writers during the past century. It was recorded in France as early as 1827. Stewart in 1910 described sporadic cases of "mosaic" in New York, and Clinton in 1914 made observations in Connecticut on a similar mosaic type. It is probable that not all of these cases of variegation were due to the mosaic virus. The successful transmission of the disease by budding and grafting was accomplished by Blodgett in 1923, and there is some evidence that the so-called "variegated leaf" was transmitted by budding in France as early as 1835. Bradford and Joley confirmed the transmissibility of the disease in 1933. Mosaic has been reported from North America, Australia, England, France, Germany, Bulgaria, New Zealand, and South Africa.

The *symptoms* are confined to the foliage, consisting of polygonal spots which in spring are white but later turn to a light yellow. In many cases the mosaic appears in only one or two branches of the tree. Vein clearing, or chlorotic vein banding, has been described in some cases. In 1951 Posnette and Cropley described three "types" of the mosaic: (1) severe, (2) vein banding, and (3) mild, based on symptom expression.

Apple mosaic virus has been transmitted only by grafting and budding. Blodgett in New York recorded the natural spread of the disease by surveys made at two 5-year intervals of a number of plotted orchards. He stated that there was no evidence of insect transmission and that the spread had a tendency to follow along rows, indicating a possible relation to pruning operations.

Apple mosaic has not been recognized in nature on any other host or connected with any other known virus. Thomas in 1937 reported successful transmission by budding, grafting, or inarching to *Cotoneaster*, *Eribotrya japonica*, *Photinia*, rose, and *Sorbus pallescens*, but not to pear, quince, *Amelanchier*, haw (*Crataegus douglasii*), or *Pyracantha*. Mosaic has been found on or transmitted to a large number of apple varieties. There is some difference in response on the different varieties, but all are susceptible. Kirkpatrick reported successful transmission of apple mosaic virus to peach. The symptoms on peach are mild and fleeting. Back transmission to apple was obtained.

No marked stunting or reduction in production has been recorded, and there is no evidence that the disease is of any great economic importance at present. (See page 176 for references.)

Rubbery Wood of Apples

This peculiar disease was first noticed in the early thirties in England on the Lord Lambourne variety. It was later found on several other varieties in England and has been reported in other European countries where Lord Lambourne is grown.

The affected trees show a lack of rigidity of some or all of their branches. Limbs an inch or more in diameter can be bent with the fingers of one hand. The normal spreading habit is replaced by a weeping appearance. The internodes are shortened, growth is retarded, and in some cases the tree is greatly stunted. The fruit is normal, but yield is reduced. The rubbery symptom becomes evident during the second year of growth. Sometimes normal wood may be produced after 5 or 6 years, in which case the new growth only becomes rigid, while the drooping habit of the previous growth is maintained. The rubbery effect seems to be due to the incomplete lignification of the fibers and vessels of the wood.

The virus can be transmitted by grafting, but no agent has been found responsible for natural spread. The disease symptoms appear in varieties other than Lord Lambourne when these are inoculated, but some varieties (Ontario and Bromley's Seedling) show no symptoms, although the virus can be transmitted to and from these by grafting. (See page 176 for references.)

Flat Limb of Apple

This is a disease primarily of the Gravenstein variety, although it has been transmitted to other varieties. Hockey in Nova Scotia claims to have found it on other varieties. It is known to occur in Australia and has been reported from the United States. It appears to be of no economic importance except on Gravenstein.

On 2- and 3-year-old wood a slight linear depression is apparent. This extends from one to several inches along the branch and approximately around one-half of its circumference. This flattening becomes pronounced with age. The affected branches are likely to be brittle and may break under a load of fruit. The bark over the flattened region appears normal, but a cross section reveals a lack of wood development in this region and increased wood development at the margins.

While this disease has been transmitted by grafting, there appears to be some doubt as to whether or not a virus is involved. Thomas reports transmission of the virus to *Pyracantha.*

Apple Rosette

In 1953 Van Katwijk described a virus disease of apples to which he gave the name "rosette." The disease was observed on Beauty of Boskop in the orchards and nurseries in two provinces of Holland (Gilderland and Zeeland). Rosettes of dwarfed leaves were described as the principal symptom. The branches were sterile where normal branches produced a good crop of fruit. Grafting to healthy trees of the same variety resulted in the development of typical symptoms. This apple rosette was given the name *Pirus* virus 6, or *Aerogenus rosettae.* Evidently this is not the same disease as described by Morris in Washington in 1923 as "apple rosette" [*Wash. Agr. Expt. Sta. Bull.,* **177**:1–30 (1923)]. He regarded this as a nutritional disorder which could be corrected by proper cultural methods.

Stony Pit of Pear

In 1939 Kienholz called attention to a disease of the Bosc pear which had been observed for a good many years previously. He proved that the disease was transmissible and was probably of virus origin.

Geographic Distribution. Stony pit has been reported from Pacific Coast states and eastern and western Canadian provinces in North America and from England and several European countries.

Economic Importance. On some varieties such as Bosc, the disease may affect as high as 70 per cent of the trees in an orchard, and Kienholz states that 10 to 80 per cent of the Bosc culls at one packing shed were attributable to stony pit. While the trees are not killed outright by the virus, much of the fruit is of no value. The limited distribution of the disease and the fact that only a few varieties seem to be susceptible would indicate that the disease is of minor or local importance.

Symptoms. On the fruit of Bosc pears "the first indication appears in from 10 to 20 days after petal fall and consists of dark green areas just under the epidermis of the fruit. The lack of growth in these areas and the rapid development of the surrounding tissues results in deeply pitted or deformed fruit at maturity." Numerous sclerenchyma cells beneath or surrounding the pitted area are produced, so that the flesh becomes woody and the fruit gnarled.

The disease may also be observed on the 2-year-old *twigs*, where a blistered condition resembling internal bark necrosis on apple is evident. On the *trunk* a peculiar ribbed appearance called "oak bark" is characteristic of the disease on Bosc after the disease has progressed for several years.

On the *leaves* the only symptom is veinlet chlorosis, that is, the appearance of narrow chlorotic bands along the veins and veinlets.

On varieties other than Bosc, the symptoms on the fruit are similar, but the oak bark symptoms on the trunk are absent or not so marked.

Transmission. The only proved method of transmission of the virus is by tissue grafting. Two or more years may elapse after grafting before the symptoms of the disease appear. In England it is reported that the disease is spreading, but the agent responsible for transmission is not known.

Varietal Susceptibility. Among known susceptible varieties are Bosc, Anjou, Beurre Clairgeau, Triomphede Vienne, and William's Duchess. Seckel, Easter, and Forelle are suspected as susceptible. Bartlett has been topworked on diseased Bosc and Anjou trees without showing any symptoms of the disease over a period of more than 5 years and with no reduction in yield. A number of other standard varieties have remained free of stony pit in regions where Bosc and Anjou are definitely diseased. Whether or not these varieties are symptomless carriers of the virus has not been determined.

Control. Graft wood should be selected from trees known to be free of the virus. Trees showing symptoms should be destroyed. Until some method of indexing trees for freedom from stony pit is worked out, there seems to be no assurance that symptomless varieties may be safely used for cion wood.

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Other Virus Diseases

Other diseases of apple which may be of virus origin are "green crinkle" reported from New Zealand and "false sting," reported from Nova Scotia, which affects the fruit only. Small depressed areas resembling insect stings appear on the young apples, and marked distortion of the fruit occurs later. It has been transmitted by grafting and is presumed of virus origin. "Chat fruit" reported from England on the Lord Lambourne variety is characterized by the upright growth of the tree, as contrasted to the normal spreading of healthy trees and the weeping habit of rubbery wood diseased trees. The fruit is small and remains green at maturity rather than the bright red of normal fruit. Luckwill and Crowdy obtained positive results by topworking chatted Lord Lambourne with healthy cions.

Atanasoff suggested in 1933 to 1934 that *bitter pit* of apple might be a virus disease, but gave no conclusive evidence to substantiate this claim.

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Diseases of Pome Fruits Incited by Nematodes

Pome fruits appear to be fairly resistant or tolerant to nematode attack. However, both apple and pear are known to be attacked by the root knot and meadow nematodes.

Root knot is common on apple in southern regions where species of *Meloidogyne* are present, especially where susceptible cover crops have served to build up a heavy population of nematodes.

Root deformations which may be confused with root knot are the galls, knots, and swellings common on apple roots caused by the woolly apple aphis (*Erisoma langerum*) and the galls caused by the crown gall bacterium.

For further information on root knot see Chap. 1 and also under Nematodes on stone fruits.

Meadow Nematode Injury. The best discussion of *Pratylenchus* injury to apple was presented by Ark and Thomas. These workers, while trying to determine the cause of "little leaf" and "rosette," found roots injured by what they identified as *Anguillulina pratensis*. Sher and Allen later reported that the nematode in question probably was *Pratylenchus penetrans* (Cobb).

On apples early symptoms appear as necrotic black or amber spots on the white rootlets, ranging from those barely visible to streaks several millimeters in length. Affected roots may be stunted and distorted. The vigor of the tree is reduced, resulting in smaller leaves and, in some cases, terminal die back.

The nematodes invade only the cortex. To what extent they alone are responsible for weakening of the trees has not been determined. It is possible that associated bacteria and fungi may play some part. It was also suggested that the wounds made by the nematodes might pave the way for the entrance of preformed toxic substances from the organisms around the roots.

For further information on the meadow nematode, see Chap. 1 and also under Nematodes on stone fruits.

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Ark, P. A., and H. E. Thomas: *Anguillulina pratensis* in relation to root injury of apple and other fruit trees. *Phytopathology,* **26**:1128–1134 (1936).

Sher, S. A., and M. W. Allen: Revision of the genus *Pratylenchus* (Nematoda: Tylenchidae). *Calif. Univ. Pubs. Zool.,* **57**:441–470 (1953).

Minor Diseases of Pome Fruits

White Root Rot. This root rot is found on a large number of fruit crops, including tree, vine, and bush fruits, and even the strawberry. In Europe it has received most attention as a grape disease, while in England and western United States it has been reported and studied more frequently on tree fruits.

This root rot is caused by an Ascomycete, *Rosellinia necatrix*, but is often referred to under the name of its imperfect stage, *Dematophora necatrix*.

The aboveground symptoms are not distinctive in that other root troubles may show similar effects. Sparse foliage, slow growth, and terminal die back of twigs are some of the features to look for. On the roots the symptoms are more distinctive. The surface of the root may be covered with a layer of cottony white growth, or this may appear when the outer layer of dead bark is removed. While strands or flat ribbons of this mycelium may be present, the thick rhizomorphs seen in the *Armillaria* root rot disease are not present. Later, the white mycelium turns brown and becomes inconspicuous. Over the dead bark or wood numerous small black sclerotia are formed. Rotting of the roots results in stunting and weakening and often the final death of the tree. The perithecia and coremia of this fungus are rarely produced in nature and probably play little part in the spread of the fungus. Infection of new roots is brought about by the growth of the mycelium through the soil or contact of the roots with old dead roots left in the soil from previously infected plants.

This root rot is often confused with the *Armillaria* root rot from which it differs in the absence of well-defined fan-shaped mats of mycelium and rhizomorphs. Also it has no "toadstool" stage. *Armillaria* does not produce the cottony overgrowth characteristic of this fungus.

A useful diagnostic feature of this fungus seen under the microscope is the presence of a pear-shaped swelling at the end of each cell in the hyphae.

In California the crab apples *Malus floribunda* and *M. toringoides* have shown considerable resistance to this root rot fungus and may be of value as rootstocks for propagating commercial susceptible varieties.

Selected References

Butler, E. J., and S. G. Jones: White root rot of apple, *Rosellinia necatrix* Prell. *In* Plant pathology, pp. 738–740. Macmillan & Co., Ltd., London, 1949.

Thomas, H. E., S. Wilhelm, and N. A. MacLean: Two root rots of fruit trees. *U.S. Dept. Agr. Yearbook,* **1953**:702–704 (1953).

Internal Bark Necrosis. In 1912 Hewitt and Truax described a papular type of bark disease occurring in the Ozark region of Arkansas. This was given the name "measles," a term generally used for the next twenty years. With the introduction of Red Delicious a type of bark injury was found to occur over a wide geographic range in the Mississippi Valley and eastward. This disease resembled that described by Hewitt and Truax and was called "Delicious measles," since this variety so often showed the disease.

For a number of years Berg in West Virginia had made a study of these papular bark diseases and in 1934 separated the "measles" and "rough bark" complex into three groups: (1) black pox caused by *Helminthosporium papulosum*, (2) internal bark necrosis, and (3) measles, which name was restricted to a condition where the pustules were smaller and more numerous than those of internal bark necrosis. In 1947 Dunegan and Isely presented evidence that the pustular type of measles as described by Hewitt and Truax was caused by leaf hopper oviposition.

Symptoms of internal bark necrosis are (1) elevations scattered over the surface of 1- or 2-year-old wood, usually with no evidence of splitting of the epidermis, (2) the presence of brown necrotic areas in the outer bark beneath the elevated pustules (Fig. 40), (3) a roughened appearance on the older twigs and limbs resembling roughening caused by scale attacks, (4) stunted growth of the limb on which the disease occurs, sometimes with terminal death of some of the smaller branches. On young trees having the disease the whole tree is dwarfed. The most characteristic symptom is the presence of the necrotic areas in the outer bark. Final death of the whole tree or individual limbs may result, although in most cases the diseased trees survive but are stunted and nonproductive.

Cause. The cause of internal bark necrosis has not been determined with certainty. Numerous experiments have established the fact that the disease is not transmissible and consequently not induced by bacteria, fungi, or viruses. Physiological studies indicate that it may be due to mineral unbalance. Berg and Clulo demonstrated that the addition of available manganese and iron compounds to the soil in which healthy Delicious trees were growing would result in the appearance of the disease. The addition of lime to acid soils where excess manganese is present, thus reducing the available manganese, proved effective in controlling the disease under experimental conditions.

Earlier, Young and Winters as a result of their experimental work on this disease were of the opinion that it was due to a lack of available boron and could be cured by the addition of boron to the soil.

Generally, the disease appears on acid soils, thus leading to the opinion by some that this condition creates a mineral unbalance in the tree which brings about the development of the disease.

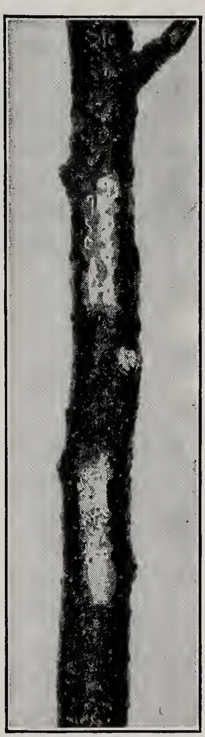

FIG. 40. Internal bark necrosis (measles) showing papules and internal necrotic islands in outer bark.

Control. Excessively acid soils should be limed to near the neutral point. The addition of boron at the rate of ⅛ to ¼ lb per year of growth of the tree is recommended, since this application will aid in the control of other diseases, such as corky core.

Selected References

Berg, A.: Black pox and other apple bark diseases commonly known as measles. *W.Va. Agr. Expt. Sta. Bull.*, **260** (1933).

——— and G. Clulo: The relation of manganese to internal bark necrosis of apple. *Science,* **104**:265–266 (1946).

Clulo, G.: The production of internal bark necrosis of apple in sand and soil cultures. *Phytopathology,* **39**:502 (1949).

Hewitt, J. L., and H. E. Truax: An unknown apple tree disease. *Ark. Agr. Expt. Sta. Bull.,* **112**:481–491 (1912).

Young, H. C., and H. E. Winters: The effect of boron, manganese, and zinc on the control of apple measles. *Ohio Agr. Expt. Sta. Bi-mo. Bull.,* **22**:147–152 (1937).

Silver Leaf of Apples. This disease is reported as prevalent on the apple in North America, extending across southern Canada and northern United States. It has not received much attention on this host, and its economic importance is not well established. It was found to be especially prevalent in Washington following years of severe winter weather. That may explain the northern occurrence of the disease in contrast to few reports from southern areas.

For a full description of the disease see Silver Leaf of stone fruits.

Selected Reference

Hord, H. H. V., and R. Sprague: Silver leaf disease of apple trees in Washington. *Wash. Agr. Expt. Sta. Circ.,* **119** (1950). (Bibliography.)

Black Root Rot of Apples. A root rot of apple, differing in a number of respects from previously described root diseases, was observed around 1913 to be prevalent in southeastern United States. This disease was studied by Wolf and Cromwell in North Carolina and reported on in 1917. In the same year Fromme and Thomas published a detailed account of the disease based on their investigations in Virginia. Later it was found to be prevalent across the belt of the eastern and central states. The cause of the disease was found to be a *Xylaria,* closely related to *Xylaria digitata* Ell. and Eve., but was renamed *Xylaria mali* by Fromme. (The name *Xylaria digitata* had been applied to a European species differing from the American species described by Ellis and Everhart and hence was invalid.)

The term "black root rot" describes one of the characteristic symptoms of the disease. The main roots of the infected trees show a black incrustation, sometimes in scattered patches but often covering the entire root for a distance of a foot or more. The wood of the root is punky and breaks easily. Infection usually starts near the crown of the tree on the larger roots but may occur some distance from the trunk. The diseased trees show evidence of weakness for several years before they are killed, but the symptoms are much the same as for other root troubles.

The fungus produces characteristic fruiting bodies at the base of the trunk, but these are not found until the disease is well advanced. These fruiting bodies are united at the base and extend upright in fan-shaped clusters to a height of from 3 to 4 in. The individual branches, or "fingers," are black when mature, but at first are covered with a white, mealy coating (*conidial hymenium*). Later the surface is studded with small elevations representing the *perithecia* embedded in the stromata. In cross section, the perithecia are found in a black exterior zone surrounding the white, firm core of the structure. This species of *Xylaria* differs from *X. polymorpha*, commonly found on decaying wood, in the extensive branching of the stromata and the flat appearance of the individual branches. *X. polymorpha* may be found in the same orchard but is easily identified by the cylindrical, thumblike stromata.

While black root rot causes considerable loss in some areas, it is not a major disease, since in general only a few trees in an orchard are killed and it is rare in most apple-growing regions. Inoculation experiments indicate that it is difficult to bring about infection, and it is only under a combination of favorable conditions that extensive invasion of the root system occurs.

Selected Reference

Fromme, F. D.: The black root rot disease of apple. *Va. Agr. Expt. Sta. Tech. Bull.*, **34**:1–52 (1928).

Pear Rust. Caused by *Gymnosporangium sabinae* (Dicks) Wint., this is a serious disease of pears in Europe wherever the trees are grown in the neighborhood of the alternate host (*Juniperus sabina*). It does not occur where pears are grown at a distance from the savin (*J. sabina*) and consequently is of very little economic importance.

The rust on *Juniper* is perennial and resembles in many respects the quince rust (*G. clavipes*) in that spindle-shaped swellings are produced from which the telial spore horns in the form of ribbons are pushed out in the spring. The aecial sori are also similar to those of the quince rust and apple rusts. The most serious effect is on the leaves, which are so extensively infected that defoliation results. The life history of this rust is similar to that of the apple and quince rusts. It can be easily controlled by removal of the juniper trees within 500 ft of the pear trees.

Brown Rot Blossom Wilt and Spur Canker of Pome Fruits. Caused by *Sclerotinia fructigena* Aderh. and Ruhl. and *S. laxa.* (see Brown Rot of stone fruit), these diseases are responsible for frequent injury on pome fruits in England and Continental Europe. The injury is more severe than that incited by *S. fructicola* in North America on these hosts. (E. J. Butler and S. G. Jones, *in* Plant pathology, pp. 740–746. Macmillan & Co., Ltd., London, 1949.)

Blister Spot of Apples. This relatively inconspicuous disease of apples sometimes causes reduction in the quality of the fruit. It was named "blister spot" by Rose in 1916, who later (1917) described the bacterium causing the disease, naming it *Pseudomonas papulans.* A more detailed study of this bacterial spot was made by Smith, who came to the conclusion that the causal bacterium was so closely related to *Pseudomonas* (*Phytomonas*) *syringae,* a widely distributed bacterium, that he named it *Phytomonas syringae papulans* (1944).

Blister spot on apples has been reported from a number of states in the Mississippi Valley region and eastward. It is not known to occur elsewhere. The disease is erratic in that it may be severe in one year and entirely absent in subsequent years. It is rarely a conspicuous disease and is often overlooked when present.

The symptoms on the fruit appear in June as darkened or water-soaked areas around lenticels. These swell into papilla-like blisters, first light in color but becoming darker on maturity. Later the epidermis dies and may crack loose from the surrounding healthy tissue. The surface of the blister is rarely roughened, as is the case with the wartlike lesions sometimes resulting from scab or insect injury. In nature the bacterium is not known to infect the leaves or twigs.

A description of the bacterium causing blister spot, now named *Pseudomonas syringae,* is given under Bacterial Canker of stone fruits.

Selected References

Rose, D. H.: Blister spot of apples and its relation to a disease of the apple bark. *Phytopathology,* **7**:198–208 (1917).

Smith, M. A.: Blister spot, a bacterial disease of apple. *J. Agr. Research,* **68**:269–298 (1944).

Phoma Fruit Spot of Apple and Quince Blotch. While here treated as of minor importance, this disease may cause extensive losses in some years where it commonly occurs. This fruit spot has been referred to under a number of different common names. It was called "Brooks fruit spot" because of the early extensive investigations of Charles Brooks in New Hampshire; in addition, "*Phoma* fruit spot," "*Cylindrosporium* fruit spot," "Quince blotch," and "*Mycosphaerella* fruit spot" are names used by various authors.

History. Fruit spot on apples first received attention by Brooks in New Hampshire in 1908. Later he reported that a fruit blotch on quince described by Halsted in New Jersey in 1892 was caused by the same fungus. In 1926 Walton and Orton announced the discovery of the perfect stage and demonstrated the relationship of a *Mycosphaerella* on overwintering leaves to the fruit spot on apples by inoculation with conidia from a single ascospore culture of the fungus. Serious outbreaks of fruit spot in the eastern states between 1925 and 1928 and again in the late thirties resulted in renewed research and, consequently, in a better understanding of the disease cycle and improved methods of control.

Loss results from the unsightly appearance of the spotted fruit. If the infection is at all severe, a large part of the fruit is worthless or is graded out as culls. Extensive rotting is not common when the fruit is kept in cold storage, but in common storage the pitting and browning of the invaded tissue becomes progressively worse. Cases are on record where 50 to 90 per cent of the fruit was rendered unfit for marketing by this disease. However, it is very erratic in its appearance and severity, so that over a period of years the loss is not great.

Geographic Distribution. This fruit spot is most prevalent in northeastern United States and southward to New Jersey and West Virginia. Westward it has been reported as common in Ohio and is occasionally encountered in Indiana, Illinois, Michigan, Missouri, and Iowa. It is difficult to account for its rare occurrence in the apple-growing regions west of Ohio since the perfect stage is always abundant on old leaves in this region.

Symptoms. The spots usually first appear on the fruit sometime during July or early August. The spots are deeper red on the colored face of the fruit and darker green on the lighter surface. They may be somewhat scattered but are usually more abundant on the blossom end. Later the spots may become slightly sunken and the tissue beneath is browned, but there is no extensive rotting. The spots on quince are much more extensive and are referred to as blotch.

Causal Organism. The fungus causing this leaf spot was known as *Phoma pomi* or *Cylindrosporium pomi* until the discovery of the perfect stage, after which it was known as *Mycosphaerella pomi* (Pass.) Lindau.[1] The *Phoma spores* are quite small, measuring only about 1 by 3 μ. They are one-celled and hyaline. While *pycnidia* are produced on the fruit spots, they rarely contain mature conidia during the season but are sometimes found in fruit in common storage. The *perithecia* of *Mycosphaerella pomi* are formed during the fall and winter on the surface of fallen leaves. They occur in groups on both surfaces of the leaf, often so crowded as to appear as black spots. They resemble the perithecia of *Venturia inaequalis* but are smaller and more superficial. They are globular, shiny black, and are 70 to 100 μ in diameter. The *asci* remain in a tight cluster when the perithecia are crushed. They measure 8 to 10 by 40 to 60 μ. The two-celled, hyaline *ascospores* average 3.5 by 19 μ.

Disease Cycle. Little information is available as to the source of primary infection. Before the discovery of the perfect stage it was postulated that the conidia formed in the overwintering fallen fruit were the source of infection.

Martin in New Jersey found that infection may occur as early as the first of June. Since the ascospores in the fallen leaves are discharged later than those of *Venturia inaequalis*, it is evident that ascosporic inoculation could account for initiation of infection, and probably for all infection, since no conidia are produced on the fruit during the remainder of the season. This is further supported by the fact that in Ohio, Young and his coworkers succeeded in getting infection by inoculating fruit with ascospores from fallen leaves. Discharge of ascospores continues during the early summer months when wet periods prevail. The infection period tapers off as the season progresses. It is not known how the fungus gets established on the fallen leaves since there is no evidence of the disease on leaves or twigs.

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[1] Class Ascomycetes, Order Sphaeriales, Family Mycosphaerellaceae.

Control. Copper sprays have given excellent control of this fruit spot. Sulfur sprays appear to be very ineffective. Contrary to earlier recommendations, spraying applications should start about 15 to 17 days after petal fall. These should be continued at about 10-day intervals through June. Ferbam at 1 to 1.5 lb to 100 gal applied 17, 27, and 37 days after petal fall also gives excellent control.

Selected References

Brooks, C., and C. A. Black: Apple fruit spot and quince blotch. *Phytopathology,* **2**:63–72 (1912).

Martin, W. H.: The Phoma fruit spot of apple. *N.J. State Hort. Soc. Proc.,* **1929**: 57–66 (1930).

Walton, R. C., and C. R. Orton: The perfect stage of *Cylindrosporium pomi. Science,* **63**:236 (1926).

Botryosphaeria Canker and Fruit Rot of Apple.[1] In 1919 Putterill described a "new apple tree canker" occurring in South Africa and determined the cause to be a *Botryosphaeria,* which he named *B. mali.* Inoculated into apple fruit, it caused a rot. Fenner, while investigating black rot of apples in eastern United States, reported in 1925 that isolations from a number of sources of presumably black rotted fruit yielded cultures in many instances of a *Dothiorella* similar to that causing currant cane blight. Inoculations into currants proved that a blight identical to that produced by *B. ribis* resulted. Fenner was of the opinion that this rot had been confused with black rot by earlier workers and that it was widely distributed in the eastern United States. He also believed that the *Botryosphaeria* described by Putterill from South Africa was *B. ribis,* although differing slightly in some morphological details. In 1924 Shear, Stevens, and Wilcox reported *B. ribis* as occurring on apples as well as on currant and a number of other hosts. Birmingham, in this same year, reported on a canker of apple trees in Australia which he ascribed to a *Dothiorella,* which Fenner found to be identical with his cultures from rotted apples. For the next twenty-five years very little attention was given to this disease by fruit pathologists. Between 1950 and 1954 Shay,[1] in Indiana, found the canker phase causing extensive killing of large limbs of Gallia Beauty, Golden Delicious, and Rome. In some cases entire trees of Gallia Beauty and Rome were killed. The fruit-rotting phase of the disease was especially severe in 1951 on fruit before and after harvest in certain orchards in southwestern Indiana. It appears, therefore, that this disease has a wide distribution and may cause extensive damage to susceptible varieties under certain climatic conditions.

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[1] Much of the information here reported has not been published, but was kindly furnished by Ralph Shay of the Indiana Agricultural Experiment Station.

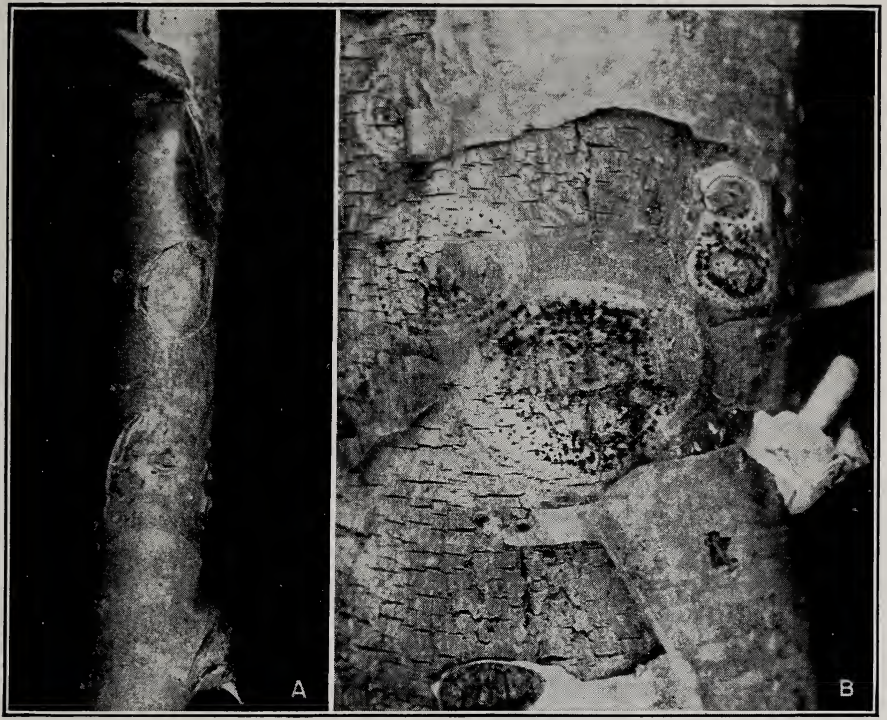

Fig. 41. *Botryosphaeria* canker on apple. At left young lesion on twig; at right fruiting bodies beneath papery outer bark on older limb.

The *symptoms* of the disease on apple twigs and limbs are distinctive. The first evidence of the disease on twigs is the presence of small circular spots. These become depressed, and often in the first infection season a watery exudate appears on the bark over and around the spot (Fig. 41). By the following spring, a few pimplelike elevations appear on the smooth surface of the lesion as a result of the production of pycnidia and stromata beneath the outer layer of bark. On older limbs young cankers may exude liquid in midsummer and become quite large. Under conditions favorable for severe development of the disease, large limbs may be girdled by fusion of cankers. A characteristic symptom is the easy sloughing off of the papery outer covering of the bark and the slimy character of the underlying tissue. On this exposed surface, rings of fruiting bodies of the fungus are evident (Fig. 42). The disease can be spotted at a distance by the dying of scattered limbs in the trees of susceptible varieties.

On the fruit two types of rot are encountered, one an external rot and the other apparently having an internal origin. The external rot resembles black rot in some respects, as stated by Fenner, but the lesions can easily be mistaken for those of bitter rot. These lesions are circular in outline, later becoming slightly depressed. The rotted flesh is cup-shaped rather than conical as in bitter rot, and the tissue is mushy rather than firm as in black rot. No fruiting bodies are evident on the surface. The second type of fruit rot is that which develops in some seasons after the fruit is harvested. Apples with no sign of external lesions may break down rapidly with a soft rot when left at room temperatures. Affected apples of red color become bleached, giving rise to the common name of "white rot." This type of rot seems to be from an internal source, the exact origin of which has not been determined.

The *causal organism* responsible for both canker and rot is *Botryosphaeria ribis,* the imperfect stage of which is a *Dothiorella.* It has not been fully established whether only the chromogenic strain is involved as is the case in currant cane blight. Both the nonchromogenic and the chromogenic strains were isolated from rotting apples by Fenner. A full description of this fungus is given under Currant Cane Blight and need not be repeated here.

Numerous *hosts* of this fungus have been reported. A serious canker disease of roses is caused by this same fungus.

The *disease cycle* is imperfectly understood. The perfect stage of *B. ribis* is commonly found on fire blighted branches of apple, often intermingled with *Physalospora obtusa.* In these cankered areas as well as on the cankers caused by *Botryosphaeria,* the spores of the imperfect stage are nearly always present, so there seem to be abundant sources of inoculum throughout the year. It appears that summer drought may play a role in conditioning trees and fruit for bringing about infection. Shay found that severe infection occurs on inoculated trees in the greenhouse only under induced drought conditions. External fruit infections in Illinois and Indiana were especially severe in a season when Golden Delicious were injured on the exposed areas by high temperatures. Thus it appears that extensive damage is to be expected only under conditions where the trees are subjected to abnormal weather conditions at a critical time for infection. Complete rotting of the fruit is usually delayed until near the ripening period.

Varietal susceptibility is very marked. Rome, Gallia Beauty, Transparent, Duchess, and Golden Delicious are especially susceptible. In an orchard in southeastern Illinois severe limb killing of Rome occurred in

1954, while neighboring trees of Jonathan, Red Delicious, and a number of other varieties were entirely free of cankers.

While here treated as a minor disease, it may be very important in some regions, although past experience indicates that over a period of years it has caused little loss.

Selected References

Fenner, E. A.: A soft rot of apples caused by *Botryosphaeria ribis*. *Phytopathology,* **15**:230–234 (1925).

Putterill, V. A.: A new apple tree canker. *So. African J. Sci.,* **16**:258–272 (1919).

Shay, J. R., and W. R. Witterly: *Botryosphaeria* canker of apple. (Abstr.), *Phytopathology,* **44**:505 (1954).

See further references under Currant Cane Blight.

Briefly Noted Pome Fruit Diseases of Minor Importance

In addition to the minor diseases previously described which warrant an abbreviated description, the following list is intended as a guide to the reader who might encounter some of these comparatively rare diseases. For each disease a citation is given either to the original description or to a later clarifying article. A brief statement indicates the general character of the disease.

Rough Bark (*Phomopsis mali* Robt.). This is described as causing a rough bark condition on Yellow Newtown apples in Virginia. [J. W. Roberts, *U.S. Dept. Agr. Bur. Plant Ind. Bull.,* **280** (1913).]

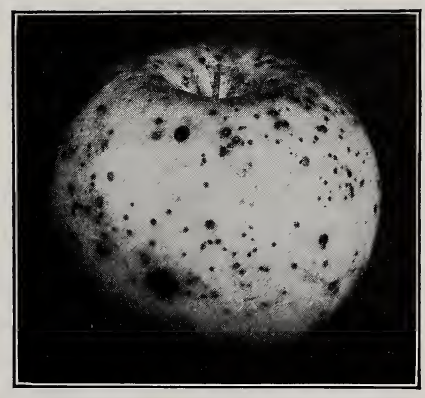

Fig. 42. Black pox (*Helminthosporium*) on apple fruit.

Black Pox (*Helminthosporium papulosum* Berg). This is a pustular bark disease similar to and confused with apple measles (see Internal Bark Necrosis). On bark it causes blackish papules which later give rise to a pitted or scaly bark condition. On fruit it causes small sunken spots (Fig. 42). It occurs on apples and pears over the eastern United States, west to Missouri. [A. Berg, Black pox and other bark diseases commonly known as measles. *W. Va. Agr. Expt. Sta. Bull.,* **260** (1934). (Bibliography.)]

Alternaria Leaf Spot (*Alternaria mali* Robt.). This is described as a weak parasite capable of enlarging injured spots on apple leaves. [J. W. Roberts, Experiments on apple leaf spot fungi. *J. Agr. Research,* **2**:57–66 (1914).]

Hypochnus, or Fisheye Rot (*Corticium centrifugum* Butl.). This often follows scab infections, causing a dry spongy rot. It is generally distributed in the United States. [L. F. Butler, *Corticium centrifugum,* a heterothallic pathogen of apples. *J. Agr. Research,* **41**:269–294 (1930). (Bibliography.)]

Pink Rot [*Cephalothecium roseum* (Fries) Cda.]. This is commonly found on scab lesions, causing a storage rot. Under damp conditions it may appear on fruit in orchards near harvesttime. It is generally distributed. [H. J. Eustace, A destructive apple rot following scab. *N.Y. (Geneva) Agr. Expt. Sta. Bull.,* **227**:367–389 (1902).]

Alternaria Core Rot (*Alternaria tenuis* Nees.)? This is commonly found as a dark growth over the seed and carpel walls of apples such as Delicious in which the calyx tube remains open. In some seasons 50 per cent of storage apples of this variety show the presence of this fungus, but rotting of flesh is not common. It is also supposed to cause blossom end rot. It is reported also on pear and is common on certain varieties. [B. O. Longyear, A new apple rot. *Colo. Agr. Expt. Sta. Bull.,* **105** (1905).]

Sporotrichum Fruit Rot (*Sporotrichum malorum* Kidd & Beaumont). This is characterized by circular or more or less irregular in outline, slightly sunken, light brown shallow spots which by coalescence of numerous spots involve large areas of fruit. It is reported from England, New Zealand, and the United States (Indiana). [M. W. Gardner, *Sporotrichum* fruit spot and surface rot of apples. *Phytopathology,* **19**:443–452 (1929).]

Storage-apple Rots. Some investigations have been made in an effort to determine (1) what fungi could be isolated from rotting apples and (2) what fungi will cause rotting when inoculated into sound apples. Heald and Ruehle succeeded in causing rots in apples by inoculation of over 40 species of fungi which they had isolated from apples. [F. D. Heald and G. D. Ruehle, The rots of apples in cold storage. *Wash. Agr. Expt. Sta. Bull.,* **253** (1931).] In England, Kidd and Beaumont in 1924 listed 51 species of fungi which had been reported by previous investigators on stored apples and described the rots they produced. This list included some of the well-known rot-producing fungi described in this text. [M. N. Kidd and A. Beaumont, Apple rot fungi in storage. *Trans. Brit. Mycol. Soc.,* **10**:98–118 (1924).]

CHAPTER 6

DISEASES OF DRUPE OR STONE FRUITS

Unlike the apple and pear, the edible fleshy part of the drupe fruit is derived from the pistil. Usually only one ovule develops, which becomes the kernel inside the stony "seed." The "stone," or pit, is formed from the inner wall of the ovary. Drupes are often called "stone fruits" because of this hard woody pit. The calyx cup, or "shuck," withers and is pushed off by the enlarging fruit. Peaches, plums, apricots, almonds, and cherries are the commercial fruits belonging in this group. Nectarines are fuzzless peaches, while prunes are varieties of plums.

All of the drupe fruits of commercial importance were introduced into North America from Europe or Asia. Wild species of plums and cherries were found by the early settlers in North America, but none of these have been utilized for commercial production. Peaches probably originated in southern China, plums from China and southwest Asia, and cherries from Asia and possibly southern Europe. A number of distinct species of plums and cherries are recognized, but all peach varieties are included in the single species *Prunus persica* and apricot varieties in the species *P. armeniaca.*

Drupe fruits are very widely distributed throughout the world. Peaches, almonds, and apricots thrive best in the warmer regions of the temperate zone, while plums and cherries do best in the cooler regions.

A number of serious fungus and bacterial diseases of drupe fruits are common to the peach, plum, and cherry groups, and for this reason these will be treated under the host on which they are of greatest importance. A large number of virus diseases attack the stone fruits, many of which have been discovered within recent years.

Brown Rot of Stone Fruits

Brown rot is especially destructive to stone fruits, but it also attacks other fruits. In this treatment emphasis will be placed on the disease as it occurs on stone fruits. The fruit rot has also been called gray mold and ripe rot. On the blossoms it is commonly called "blossom blight," and on the twigs, "twig blight." When the branches are invaded the resulting lesion is called "brown rot canker." In this treatment, the term "brown rot" will include all stone fruit rots caused by species of *Sclerotinia.*

History. The early history of brown rot as a disease is "lost in antiquity." This is due in part to the fact that rotting of peaches apparently was accepted as a natural process, especially as fruit brown rot ordinarily is first observed by laymen near or during the time of ripening. Thus, many of the early horticultural writers who were evidently quite familiar with brown rot mention it casually, if at all, or associate it directly with the plum curculio injury. The appearance of the "mold" on the rotting peaches was evidently considered secondary. The European brown rot (*Sclerotinia fructigena*) was recognized by a botanist as early as 1796. The American brown rot was mentioned as early as 1807. The recent discovery that the two European species of the brown rot fungus are rare in America and that the American species is equally rare in Europe leads to the conclusion that the American brown rot was probably native to the wild plums of this region and, like the curculio, found a congenial host in the cultivated peaches.

The first scientific statement in regard to the fungus in America was by Schweinitz in 1822, who mentions the fungus as common on peach. Riley, an entomologist, in 1869 mentions the disease as being common in Missouri and Illinois following the attack of plum curculio.

Brown rot was apparently not considered a serious disease in most sections of Europe, and while the fungus was described in the latter part of the eighteenth century little attention was given to the disease. In America, however, conditions in the great peach belt of the southeastern section of the United States were unusually favorable for the development of the disease, and as early as 1852 White of Athens, Georgia, writes, "But our greatest obstacle in the culture of the plum is its tendency, in common with several varieties of the peach, nectarine, and grape, to rot before maturing."

While Schweinitz collected the brown rot fungus in 1822, it was not until 1881 that any attention was given by botanists to the relation of the fungus to the disease. In this year Peck described the fungus and demonstrated its pathogenicity by inoculation. In Europe the first account of brown rot from an economic standpoint was by von Thümen in 1875. Arthur in 1886 called attention to the blossom and leaf blight of cherry.

In 1902 Norton found, in Maryland, the perfect stage of the brown rot organism on peach and plum mummies and proved its connection by inoculation. For many years this was supposed to be the first record of the perfect stage. It was later discovered that Eugene Rau of Bethlehem, Pennsylvania, had found this stage on the peach in 1883 and had sent specimens to Winter in Europe, who named it *Ciboria fructicola.*

It was assumed from the time of Schweinitz until recently that the brown rot organism of America was the same as one of the two species occurring in Europe. Between 1913 and 1932 a number of publications were issued in which the relations between the various species of brown rot were discussed. These relationships will be treated under Causal Organisms.

Quaintance in 1900 reported the results of his study on the brown rot in Georgia in which attention was given to the economic importance of the disease in this favorable region. At that time the only fungicide commonly used was Bordeaux mixture, and Quaintance called attention to the serious injury resulting from the use of this mixture on peaches. With the introduction of lime sulfur as a summer spray, attempts were made to control brown rot, but again it was found that extensive burning and defoliation would result under certain weather conditions. In 1907, Scott introduced self-boiled lime and sulfur as a safe fungicide for peaches. For many years this was used successfully in the control of peach brown rot. With the introduction of finely ground sulfur as a dust about 1912, this material was also generally used for brown rot control. Various types of "wettable" sulfur were developed for peach liquid spraying. Of these the one most widely used for some time was the New Jersey Dry Mix. Commercial companies soon produced a number of "wettable" sulfurs which could be substituted for the New Jersey Dry Mix.

Contributions to the life history of the brown rot organism were made by numerous investigators in America, Europe, and Australia. Arthur (1886), Galloway (1889), and Smith (1889–1891) called special attention to the blossom blight of stone fruit. Chester in 1893 inoculated peach blossoms and produced typical blossom blight. The canker phase of brown rot was given special attention by Jehle, who reported in 1913 the results of observations on artificial and natural infection in twigs and limbs of the peach. He also called attention to the perennial nature of some of these cankers. Roberts and Dunegan in 1932 published a monographic bulletin on the American brown rot.

Geographic Distribution. Brown rot is found in all countries where the stone fruits are grown, but is of little importance in regions of low rainfall during the ripening period of the fruit. In America most damage occurs in the southeastern peach-growing area and along the Atlantic seaboard, from New Jersey southward. Severe epidemics are common in other regions of the eastern United States under favorable conditions for the development of the fungus. The relative prevalence of plum curculio and the oriental fruit moth are also important factors in the seasonal severity of the disease. In the western area of the United States brown rot is of importance only in those sections where moist conditions prevail, such as in the region of San Francisco Bay. According to Heald it is rare in the Pacific northwest in the dry regions east of the Cascades, but is serious on cherries, prunes, and peaches in the humid sections of western Oregon and Washington. It is also reported as serious during certain seasons in New Zealand, Australia, Japan, and in some sections of South Africa.

Economic Importance. The greatest loss from brown rot occurs directly as a result of rotting in the orchard, in transit, and on the market. Ordinarily stone fruits are not stored for any length of time, and consequently storage loss is not great. Reduction of yield also occurs as a result of blossom blight, but the more serious aspect of this is the production of twig cankers, thus increasing the possibility of fruit rot later in the season. Cankers, unless very extensive, do not entail serious losses in the crop, since it is a common practice to thin out branches in pruning and the selection of cankered branches in this process is not likely to seriously affect the total production. Cankers, like blossom blight, may cause indirect loss by furnishing inoculum for fruit infections.

The complete destruction of the fruit by brown rot with only a minor loss from other causes permits a fairly close estimate of actual loss. Before a successful fungicide had been found it was not unusual for growers to report from 50 to 75 per cent loss of the crop in the orchard in a wet season, and the entire crop was often destroyed before it reached the market.

For peaches alone the estimated average annual reduction in yield from 1917 to 1925, inclusive, was 3,200,000 bushels. Added to this should be the extensive loss to cherries, plums, prunes, apricots, and nectarines. The loss in transit is often from 5 to 25 per cent, according to the figures of government market inspectors. While exact figures of loss in the retail markets are not available, one purchasing peaches cannot help but be impressed with the seriousness of this loss.

In England, loss from brown rot as a result of twig blight is regarded as one of the most serious aspects of the disease.

Symptoms. Since the symptoms of brown rot are much the same regardless of the particular species concerned, the following description will be based mainly on the disease as it occurs on stone fruits in eastern United States.

The brown rot organism causes blossom and twig blight, fruit rot, and canker. On the *blossom* it may attack any of the floral organs but is usually found first on the organs approaching senility, such as wilting stamens, mature petals, the withering stigma, and sepals (shucks). The organs attacked turn to a gray or light brown color. The fungus grows rapidly, soon involving the clustered mass of stamens with their anthers. These together with the decaying petals are soon covered with the gray conidia of the fungus if wet weather prevails. The rotting mass clings to the twig for some time and may be later pushed off by the developing young pistil, which often escapes infection. In other cases the young fruit is invaded through the pistil, and the fungus passes down into the twig where a definite oval or elliptical canker may be formed, or the twig may be completely girdled.

Cankers may arise from various types of invasion. As described above, cankers may develop on twigs or branches as a result of blossom blight.

The fungus also may grow down the fruit pedicel, following fruit rot, or down a blighted twig into the larger branch. On the twig and smaller branches the brown rot cankers are usually elliptical at first, rather definite in outline, and brown in color. They are usually sunken, but on the other hand, in cases where the underlying tissue is not completely killed, they may be hypertrophied and raised above this surrounding tissue. During rainy weather gumming is common on the cankered surface and the gray spore tufts appear scattered over the surface of the bark (Fig. 43). If the twig is not completely encircled, callus formation later occurs around the invaded area, and with the closing of this over the necrotic area a spindle-shaped enlargement is seen later in the season. A longitudinal cut through the twig shows a dead area in the wood below the callus which may be several inches in length. Necrotic areas around the bud as a result of arsenical injury are often confused with true brown rot cankers. Arsenical cankers may be distinguished by the fact that they are likely to occur around each bud, whether blossom or leaf bud, in a definite area on the twig.

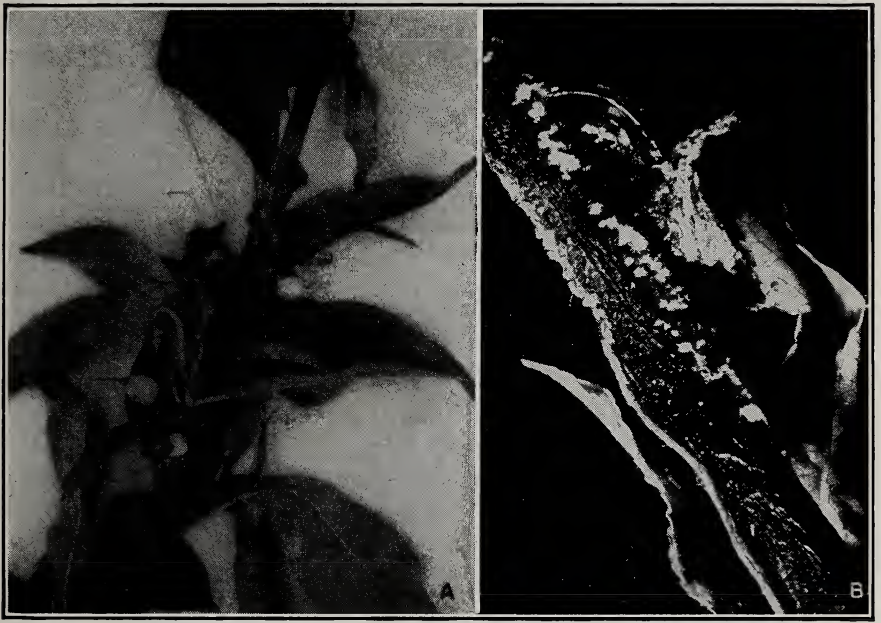

FIG. 43. Brown rot canker on peach twig resulting from blossom blight. Enlarged at right to show sporodochia.

Infection of the twigs and larger branches by invasion from rotted fruit also produces cankers somewhat similar to those described above. Invasion from the fruit is more likely to result in killing the entire branch. A type of canker frequently seen in some years is that produced by the complete girdling of the twig without killing of the distal portion. The following spring a decided swelling is seen above the girdled portion, but the leaves appear normal until about half grown, when the entire branch above the girdled area dies, after a preliminary yellowing or wilting of the leaves.

Perennial cankers have been described by various authors. These seem to be associated with weak trees in regions where winter injury is fairly common and are not commonly observed on young vigorous trees even following extensive fruit rot. In Europe, brown rot cankers of this type have been described as common on cherries, plums, and apples. These cankers as they occur on the older limbs of the peach in the United States are characterized by a copious flow of gum during certain seasons of the year. The surface is very rough and when cut into reveals a mass of old hard bark, gum masses, and remains of old callus tissue. A heavy growth of callus is formed around the edge of the canker. While these cankers may originate from brown rotted twigs or fruit, there is some doubt about the fungus being responsible for the annual killing back of the callus tissue.

Brown rot on the *fruit* becomes more noticeable as the fruit approaches maturity. The first evidence of the rot is the appearance of a small circular brown spot, frequently originating in some slight wound caused by any of several different insects, mainly curculio and stink bugs, although in some cases, especially on fully ripe peaches, no evidence of wounding may be seen. Many sucking insects feed on maturing peaches without leaving visible wounds. One to several rotten spots may appear on a single fruit, but commonly only one or two are present (Fig. 44). The rot develops very rapidly if the fruit is mature. The outline remains circular, and the color is light brown. The surface remains smooth and is not sunken. The rotted flesh is softer than the surrounding tissue but is not watery, as in the case of *Rhizopus* rot.

Sooner or later the rotted area becomes more or less covered with ash-colored tufts which break through the skin of the peach. These masses of conidia give the fruit rot a brownish gray appearance (buff yellow in the case of the European species). The time of the appearance of the fruiting pustules depends on weather conditions, on rapidity of the rot, and probably on the strain or species of the fungus involved. Often the fruit is entirely rotted before the first tufts appear, while in other cases they may appear within a day or two after the first evidence of the rot spot. Frequently the pustules are so numerous that the entire surface of the fruit is covered, but in other cases they may be few in number or arranged in rather definite concentric rings some distance apart. Ordinarily the pustules appear sooner and are more pronounced on the plum and cherry than on the peach (Fig. 45).

The fruit usually retains its form and remains attached to the tree for some time after it is completely rotted, then it either falls or, if retained on the tree, gradually dries into a firm mummy. The fallen fruit, if not badly crushed, also mummifies. During this process of drying a black encrustation of fungus tissue becomes evident which appears to replace the normal skin of the fruit. The internal flesh of the fruit becomes dry and spongy. The mummified fruit remains firmly attached to the tree, or if on the ground, it does not disintegrate as is the case with fruit rotted by other organisms. In the peach it often happens that a certain per cent of the fruit ceases growth when about the size of a normal peach seed. These stunted peaches normally drop (the so-called "June drop"), but sometimes they remain attached to the tree and are often confused with brown rot mummies during the winter months. The black encrustation characteristic of brown rot mummies is not present on these.

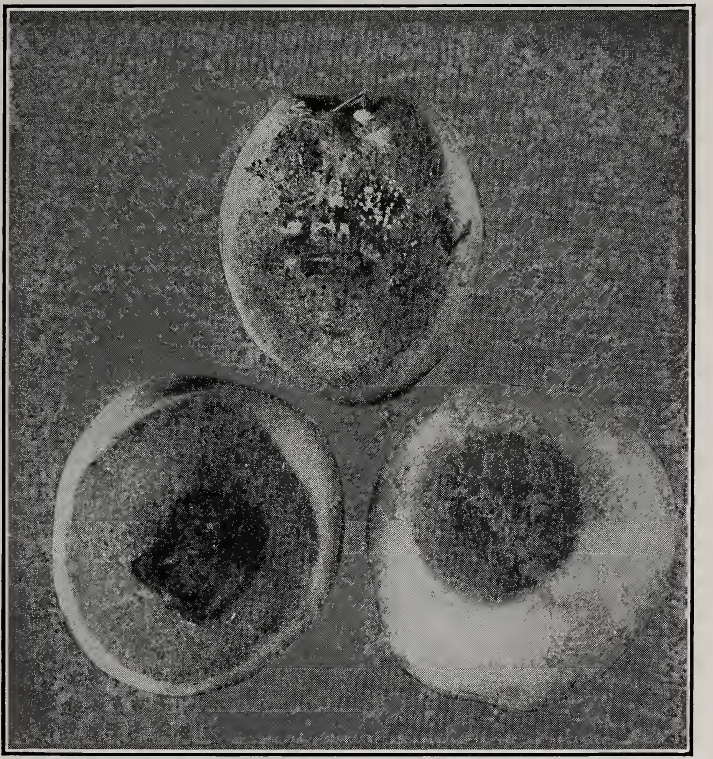

FIG. 44. Brown rot on peach showing fruiting pustules (upper) and origin from wound (lower right).

In the spring, mummies attacked by brown rot are distinguished by the presence of the black sclerotial layer. In some cases new crops of conidia are formed on these, and thus they take on a dusty gray appearance.

Twig blight, or "wither tip," as it is commonly called in England, is a symptom rather rare on peaches but common on some varieties of plums during a very wet spring. Twig blight may occur as a result of the extension of the invaded tissue at the attachment of the blossom or fruit stem to the twig, but in the typical "wither tip" it is the result of infection either on the leaves or the young stem, and from these it progresses downward, producing a blight similar in appearance to fire blight in apple. However, in peach and plum twig blight the leaves are usually a dull green in color, later turning to a light brown. In very damp weather conidial tufts and gumming may be evident.

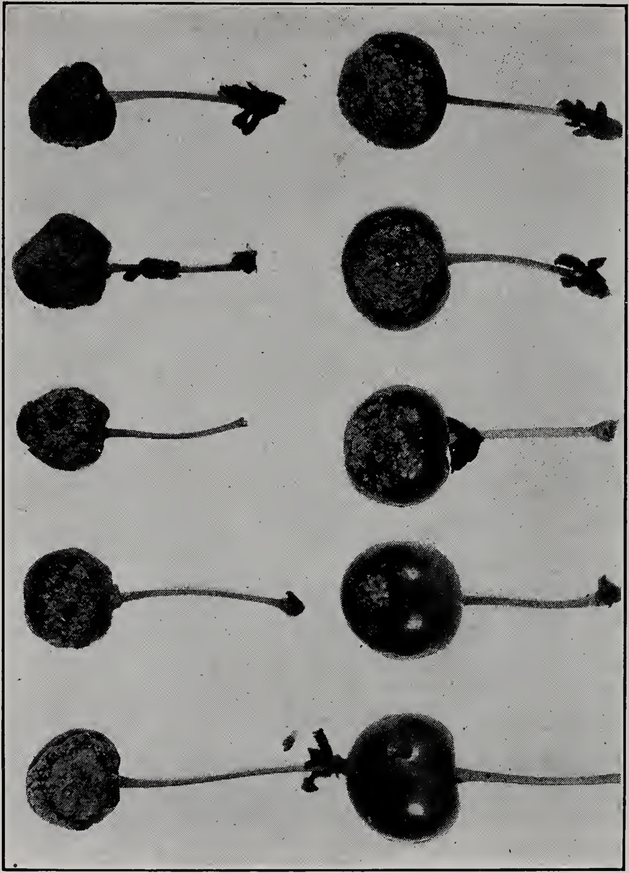

Fig. 45. Brown rot on cherry in various stages of development.

Causal Organisms.[1] Persoon, in 1796, described a fungus causing brown rot of fruit in Europe under the name *Torula fructigena,* which he later (1801) transferred to the genus *Monilia.* Kunze and Schmidt in 1817 transferred it again to the genus *Oidium.* The Persoon name of *Monilia fructigena* was generally used throughout the nineteenth century for all European as well as American brown rots of fruit, although in 1851 Bonorden described another species occurring in Europe under the name *M. cinerea,* also occurring on stone fruits.

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[1] *Sclerotinia* spp., Class Ascomycetes, Order Pezizales, Family Helotiaceae.

In America, the early mycologists seem to have accepted the European name of the fungus, since Schweinitz in 1822 labels a specimen on peach *Torula fructigena.* Ravenel specimens collected in South Carolina on peach are labeled *Oidium fructigenum.*

A collection made by Eugene Rau of Bethlehem, Pennsylvania, in 1883 and sent to Winter in Germany is of great interest from the standpoint of the taxonomist. This collection from a decayed peach contained apothecia. Unfortunately, Winter in describing this fungus named it *Ciboria fructicola.* In 1886 on the authority of Rehm, Saccardo transferred this to the genus *Sclerotinia* and listed it as *Sclerotinia fructicola* (Wint.) Rehm. It was not until 1909 that the possible connection between this fungus and brown rot was considered by mycologists. In that year Pollock suggested that the ascigerous stage of the American brown rot fungus might be that described by Winter. Again the facts concerning this collection were generally ignored by mycologists until 1924, when Roberts and Dunegan, after the establishment of the American fungus as a species distinct from the European, again called attention to this early collection.

Woronin in 1888 demonstrated the connection between a *Monilia* and a *Sclerotinia* on mountain cranberry (*Vaccinium vitis-idaea*). On the basis of this discovery Schröter in 1893 proposed the names *Sclerotinia fructigena* and *S. cinerea* for the two fruit rotting *Monilia* species. Although this was generally accepted, Schröter gave no proof that the perfect stage of these *Monilia* species was a *Sclerotinia.* He seems to have overlooked Winter's previous description of *Ciboria fructicola.*

The first definite proof of the relation between any of the brown rot *Monilia* species on stone fruit was that of Norton in 1902. He found abundant apothecia developing on peach mummies in Maryland in the spring of that year and by cultural work established beyond a doubt the identity of the *Sclerotinia* apothecia and the *Monilia* form on the brown rotted fruit. Since the American *Monilia* was still regarded as *Monilia fructigena,* Norton named the fungus *Sclerotinia fructigena* (Pers.) Norton.

Three years later Aderhold and Ruhland finally discovered the apothecial stage of the European brown rot fungus *M. fructigena* and demonstrated its connection with the *Monilia* stage. But Aderhold and Ruhland also called attention to the fact that the American brown rot fungus was not *fructigena* but was probably *M. cinerea,* and Norton's species should therefore be *S. cinerea.*

Although Bonorden had described *M. cinerea* as early as 1851 and although the description of this fungus more nearly fits that of the American brown rot, American writers in general continued to use the name *S. fructigena.* Matheny (1913), Conel (1914), Bartram (1916), and Jehle (1913) all called attention to the resemblance of the American brown rot fungus to Bonorden's *M. cinerea,* and after about 1915 the name *S. cinerea* was accepted by most writers.

Wormald in England in a comparative study of the cultures of the American organism and the European *S. cinerea* came to the conclusion that the American brown rot fungus was different from that of Europe, and he called it *Sclerotinia cinerea* forma *americana*. In 1921 he found the perfect stage of *S. cinerea* in England, and although this was the first definite proof of the connection between the *Monilia* and apothecial stages of this species, he accepted Schröter's earlier suggestion and named it *S. cinerea* (Bon.) Shr. Ezekiel in 1923, confirming Wormald's results by extensive comparative studies of the European *S. cinerea* and the American form, proposed that the latter be given specific rank and so renamed it *S. Americana* (Worm.) comb. nov.

In 1924, Roberts and Dunegan, having made a very close study of the type material of the Rau collection of apothecia of *Ciboria fructicola* and being convinced that it was the apothecial stage of the *Monilia cinerea* of America, called attention again to Pollock's statements in 1909 and pointed out that the correct name of the American species should be *S. fructicola* (Wint.) Rehm.

Wormald in a series of articles between 1920 and 1930 calls attention to several strains of *S. cinerea* in England. He accepts the specific rank of the American brown rot given by Ezekiel and Norton. According to this author, *S. fructigena* (Pers.) Schr., not known in America but present in England and on the Continent, causes fruit rot of apples, pears, plums, and cherries; *S. cinerea* (Bon.) Schr. is present on apples, pears, plums, peaches, and cherries; forma *mali* is confined to Europe, producing blossom wilt and canker of apples; forma *pruni* produces fruit rot and wither tip of plums and cherries and is present in England, the Continent, and in several American localities.

Honey in 1928 proposed that all species of *Sclerotinia* that have *Monilia* as the imperfect stage be placed in a new genus *Monilinia*. Thus *S. fructicola*, which he selected as the type of this genus, would become *M. fructicola* (Wint.) Honey. Acceptance of this change has been slow, and since *Sclerotinia* is a well-known as well as a valid genus, this name will be retained.

In 1923 Barss reported a brown rot organism from Oregon which differed from the ordinary American form and proposed the name *M. oregonensis* Barss and Posey. Later this proved to be the same as *S. cinerea* forma *pruni* and has been renamed *S. laxa*.

The present status of the taxonomy of the brown rot organisms common on stone and pome fruits may be summarized as follows:

Sclerotinia fructigena (Pers.) Schr. A true European species not known in America.
Sclerotinia laxa Aderh. and Ruhl. Not common in America. Reported in a number of stone fruits in the West and on cherries in Wisconsin and Michigan.
Sclerotinia fructicola (Wint.) Rehm [*Sclerotinia Americana* (Worm.) Norton and Ezekiel]. The common brown rot of stone and pome fruits in America, Australia, and New Zealand.

Hosts. Various species of *Sclerotinia* attack all the commonly cultivated drupe and pome fruits and also many of the native species of the rose family which have fleshy fruits. Until more work is done on the separation of the species it would not be possible to give a complete list of host plants for each species with any degree of accuracy. *S. fructicola* is most destructive to the stone fruits in America. It is of minor importance on apple, pear, and quince. *S. fructigena,* on the other hand, is regarded as one of the most serious apple rots in England.

Morphology. Since the several species of *Sclerotinia* on stone fruits are so morphologically alike that they can be separated only with difficulty, the following description, based on *S. fructicola,* will serve for the other species except where the variations are noted.

The brown rot organism is an ascomycetous fungus, and as is commonly the case, the parasitic phase is the conidial, or imperfect, stage. The perfect stage appears in the spring on mummied fruit in the form of a stalked apothecium.

The *hyphae* are branched, septate, and at first hyaline. The protoplasmic content is finely granular with no distinctive characteristics. True *sclerotia* are not formed in culture, but the firm black layer on the outside of the mummied fruit is ordinarily designated as a sclerotium. This encrustation is made up of a mass of dark, thick-walled hyphal cells with a certain amount of included host tissue.

Conidial tufts, or *sporodochia,* are produced both in culture and on the host, although at times these are so closely associated as to give the appearance of a solid mass. It is claimed by some that in *S. fructicola* mycelial cushions at the base of these tufts are absent or inconspicuous, while in both *S. laxa* and *S. fructigena* they are quite definite. The color of the tufts is one of the distinguishing characters of the various species. *S. fructicola* has an ash-gray color, while *S. fructigena* is described as buff yellow.

The *conidia* are borne in chains, arising from hyphal branches which cannot be regarded as distinct conidiophores from a morphological standpoint, although the grouped arrangement of these structures indicates a specialization in their function. The conidia-producing hyphae form a nodulate chain, each nodule later rounding off by a process of constriction into a conidium.

The conidia are lemon-shaped (limoniform) or elliptical. The shape and size varies somewhat in the various species, and there is also a rather wide variation within the species depending upon the conditions under which the spores are developed and, according to some authors, on the strain of the species. Average measurements for the three species are, approximately: *S. fructicola,* 14 by 11 μ; *S. fructigena,* 18 by 11.5 μ; *S. laxa,* 14 by 10 μ. The measurements may vary from the average as much as 10 μ in length and 6 in width.

Microconidia are commonly formed both in culture and in nature. They appear as viscid, cream-colored to tan masses in culture and on mummied fruit. They are produced on flask-shaped conidiophores with an average length of 6 μ and an average width of 2 μ. The microconidia are borne in long chains, are globose, hyaline, 2 to 3 μ in diameter, with a single, distinct refractive globule, probably an oil drop, in each spore. The conditions under which microconidia are produced and their function are not known, but they may be male cells. Various types of *chlamydospores,* or thick-walled cells, are also formed by the brown rot organism.

Apothecia. All the fruit rotting *Sclerotinias* form apothecia which are alike in structure. They arise from the mummied fruit, which has become partially or entirely embedded in the soil or debris (Fig. 46). One to twenty or more may arise from a single mummy. The apothecia are stalked, the length of the stipe depending upon the depth of the mummy. When the apothecium arises near the ground line, the stipe may be almost absent, while if the mummy is completely buried, it may be 2 in. in length. The "cup" of the apothecium develops rapidly after the tip of the structure appears above the ground. At first it is slightly swollen, later becoming globose with a small opening in the center of the upper surface. This gradually enlarges, but the upper edge of the cup remains incurved until the apothecium is more fully developed. When mature the apothecium is funnel-formed, with the edges slightly recurved. Later, by further expansion, it may be disk-shaped. Apothecia ordinarily measure from ⅛ to ½ in. across and are light brown to buff in color. The disk is rather thin and frequently splits at the edge as it expands.

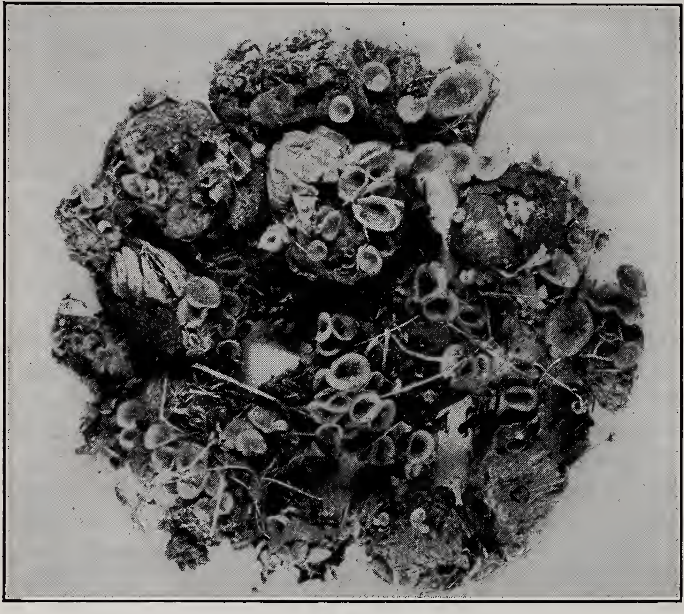

FIG. 46. Brown rot apothecia arising from mummied fruit on ground.

The *asci* are borne in a compact hymenium interspersed with filiform paraphyses which are slightly longer than the asci. The asci are cylindrical to narrowly clavate, with a thin wall and an indistinct pore at the apex. They are on the average about 150 by 10 μ. The eight single-celled hyaline *ascospores* are elliptical in outline and are arranged in a single row, the spores having a rather definite oblique slant (Fig. 47). The measurements of the ascospores are much the same in all the fruit rotting species, 6 to 15 by 4 to 8 μ.

In culture the three fruit rotting species can be distinguished by certain rather constant differences, although the variations within the species may be so marked as to lead to confusion. *S. fructigena* forms larger pustules, yellowish in color, and has larger conidia than *S. fructicola* or *S. laxa*. *S. laxa* grows more slowly on most media than does *S. fructicola*. Zonation is also more common, and the edge of the culture is lobed. Rotting of the fruit is more rapid with *S. fructicola* than with *S. laxa* and conidia are more likely to develop early. The characteristic ash-gray color distinguishes these two species from *S. fructigena.*

Disease Cycle. The source of primary infection of brown rot in the spring varies with climatic as well as other conditions. The possible sources are (1) overwintering of conidia on mummied fruit on the tree or on the ground, (2) conidia produced in the spring on mummied fruit, (3) ascospores produced in apothecia on mummied fruit on the ground, and (4) conidia on cankers produced the preceding year. One or several of these sources may account for blossom blight, which in turn may produce cankers on which the conidia for fruit infection later in the season are developed. This seems to be the most common sequence of events in the region where blossom blight is common. In certain sections of the country, as in the Middle West, blossom blight on the standard commercial varieties is comparatively rare, and even in a season unfavorable for blossom blight, brown rot of the fruit at maturity may be extremely severe. Blossom blight usually results from infections of stamens and petals at early bloom stage.

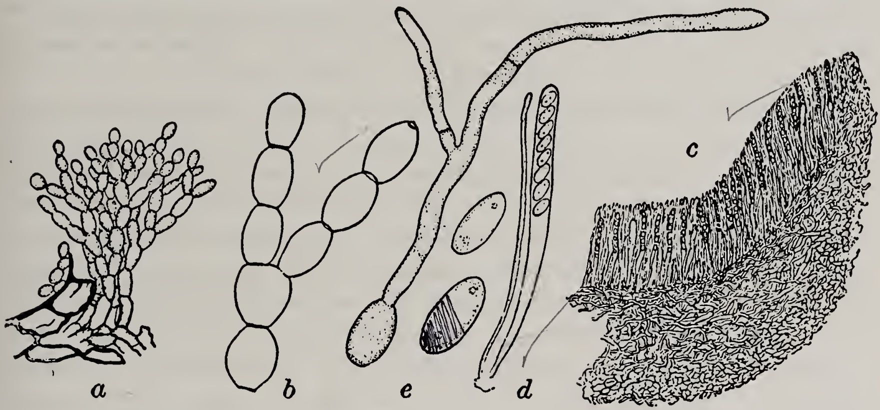

FIG. 47. *Sclerotinia fructicola. a,* conidiophores; *b,* chain of conidia; *c,* section of apothecium with asci; *d,* asci containing ascospores and a paraphysis; *e,* ascospores and a germinating conidium. (*After Duggar.*)

The ascospores are forcibly discharged, the discharge being brought about by changes in humidity and slight disturbances. The simultaneous discharge of large numbers of ascospores may produce a "puff" visible to the naked eye. These spores are carried by currents of air for some distance, and when the apothecia are numerous, may well account for blossom infection. However, in cultivated orchards where the fruit is kept cleaned up, apothecia are extremely hard to find. They may be found abundantly in neighboring wild plum thickets or under seedling peach trees in the fence rows or neighboring wood lots.

The apothecia soon disintegrate, and since the ascospores have a relatively short period of viability, there is little possibility of fruit infection from these.

Conidia produced on mummied fruit may survive the winter and cause infection the following season. The conidia formed late in the season are thicker-walled and more resistant than those produced during the early summer. These may account for either blossom blight or fruit rot. Conidia may also be produced in the spring on mummies on the tree, but, according to Roberts and Dunegan, not on those on the ground. This is probably the most common source of primary infection since the mummies on the tree are in a good position to cause blossom infection.

Conidia as well as ascospores are mainly wind-borne. They are easily detached and may be seen drifting in clouds from a rotted fruit which is disturbed. They are not easily wet but are inclined to float on a drop of water and settle around the edge of the drop as it dries.

Undoubtedly, the most important phase in the life history of the brown rot organism from a commercial standpoint, at least, is the source of fruit infection. Infection of unwounded fruit is rare even when it approaches maturity. During the growth of the fruit, however, wounds or insect punctures open up courts of infection, and conidia from the blossom blight cankers may cause sufficient rotting to carry the inoculum through the early-growth period of the fruit. As the fruit approaches maturity the chances for wounding and subsequent infection are increased. Insect and twig punctures, limb rubs, splitting of the fruit at the stem end, and hail injuries are responsible for the rapid build-up of rotting as the season progresses.

Insects are very important in the production of brown rot epidemics. This fact has become so evident that it is generally stated that in a year when the plum curculio and oriental fruit moth are scarce, brown rot is not likely to be serious, regardless of favorable weather conditions for its development. While insects evidently carry spores on their bodies, they are not important agents of dissemination, but the wounds which they produce in the fruit serve as courts of infection.

In transit, peaches often are rotted to the extent that they are of little value upon arrival. In most cases the cause of this rot can be traced to the infections which have occurred at the time of packing or to the numerous wounds produced by handling the fruit in the process of picking and packing.

In conclusion it may be stated that brown rot spores are almost omnipresent during the ripening period of the fruit and that infection is certain to occur if weather conditions are favorable and if courts of infection through wounding have been provided.

Relation to Environment. Brown rot of stone fruit is greatly influenced by weather conditions, especially by the rainfall at critical periods. The temperature relations are not so important since germination of brown rot spores will occur over a wide range. The optimum temperature for germination is between 75 and 80°F, which is near the summer temperature during cloudy weather and at night, when infection is likely to occur. Brown rot spores will germinate at as low as 32°F, but growth at this temperature is very slow. In transit, it has been found that rot develops very slowly where temperatures of 40 to 45°F are maintained in the car.

Under favorable moisture and temperature conditions the conidia germinate very rapidly and infection may occur within a few hours. At low temperatures the incubation period is decidedly increased. The rot in the fruit, after infection, spreads very rapidly, and the rate of spread is determined by temperature and maturity of the fruit rather than moisture conditions.

Control. *Sanitation and Orchard Management.* In regions where blossom or twig blight occurs, the removal of mummied fruit from the tree and orchard is of importance. It is not possible to control brown rot by this method alone, and its exact relation to control has never been accurately determined. The prevalence of wild plums, seedling peaches, and neglected farm orchards of stone fruit as well as pears and apples in the neighborhood of commercial orchards complicates control by sanitation. Cultivation is of importance mainly in reducing the chances for the formation of apothecia and reducing the curculio population of the orchard. Apothecia ordinarily develop only in shaded places protected from the direct rays of the sun. Such situations are rare in a properly cultivated orchard. Cultivation does not ordinarily bury the mummies to a depth sufficient to prevent the formation of apothecia, and in fact may place the mummies in a more favorable position for their production. The value of cultivation lies in the fact that the mummies are more exposed to drying effects when the grass and weeds are removed.

Proper pruning aids in control in that it allows free circulation of air and more rapid drying of both foliage and soil during rainy periods. It also reduces the chance of twig punctures of the fruit.

Plums as well as peaches often rot as a result of contact with one another. Thinning is advocated not only as a good cultural practice but also to reduce the chances of brown rot infection.

Removal of fruit infected with brown rot is of great importance during the later part of the season. This should be done before the production of the conidia on the rotted spots and before the rot has had a chance to enter the stem, thus preventing cankers.

Varietal Considerations. Fruit growers have learned by experience that it is unprofitable to grow certain varieties which are especially susceptible to brown rot. While a number of factors are involved in susceptibility, the total effect may be summed up by stating that those varieties with a thin skin and soft flesh are more likely to suffer from brown rot, while those which ripen evenly, do not bruise easily, and remain firm after harvest are resistant.

Blossom blight is most likely to occur on early varieties of peaches. Those with large petals are more likely to suffer than the small-flowered varieties.

Control by the selection of resistant varieties has a definite place in the general program of production, and few commercial growers consider the use of varieties which will not stand up under transportation. Unfortunately, the more desirable varieties from the consumer's standpoint are those especially susceptible to rot and can be grown only where used in local markets.

Spraying and Dusting. No method of control has been devised which will replace spraying or dusting. Fortunately, effective and comparatively inexpensive methods of control by this means have been developed. The first efforts in the use of fungicides resulted in failure, owing to the serious injury to peach by Bordeaux mixture, although the control of brown rot of cherries and plums was more successful. Lime sulfur was also found to be injurious to peach foliage, and it was not until the development of the self-boiled lime and sulfur in 1907 that a safe fungicide for peaches was generally available to the growers. Later wettable sulfurs, both homemade and commercial, came into general use. Dusting with sulfur was also found effective on brown rot control on peaches, since the dust particles were entrapped by the hairy coating of the peach.

Where *S. laxa* is the prevalent brown rot organism, as in Pacific Coast areas, dormant eradicant sprays are applied to reduce sporodochia formation.

Some of the organic fungicides have proved more effective than wettable sulfur in controlling brown rot of peaches. Phygon applied during the early bloom stage reduces blossom blight infection and thus decreases the chance of twig canker development. Captan applied several times during the 3 weeks before harvest has given control superior to sulfur.

By far the most important phase of spraying for brown rot control is the effective control of the plum curculio. Experiments have shown that in the absence of curculio and oriental fruit moth, brown rot is rarely of any importance in the orchard unless unusually wet weather prevails at the time of harvest.

Harvesting and Transportation. Brown rot probably causes more loss in transit and on the market than in the orchard, although most of this loss is due to the rot which develops as a result of factors encountered in the field. Wounds produced during harvesting and packing are ideal courts of infection, since the fruit at this time is very susceptible and brown rot conidia are very abundant not only in the orchard but especially in the packing shed. Fingernail scratches, contact with rough containers, nails in baskets, bruising of overripe fruit, all contribute to the start of invasion, which results later in many rots in the basket.

Growers have had success in controlling brown rot in transit by careful handling of the fruit and dusting in the orchard a day or so before harvest. Prompt reduction of temperatures by precooling with stericoolers or hydrocoolers and immediate refrigeration are also essential for checking rot in transit. In modern refrigerator cars provision is made for air circulation, since it has been found that there is a marked difference between the temperature on a level with the first layer of baskets as compared with that in the third, and that consequently much more rot develops in the upper than in the lower layers.

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Bacterial Spot of Stone Fruits

Some of the common names of the disease now generally called bacterial spot are "shot hole," "bacterial shot hole," "bacteriosis," "black spot," and "bacterial canker."

History. Bacterial spot is almost certainly of American origin. This is substantiated by the fact that it was long after the disease was first described in this country before it was reported as occurring elsewhere. On the other hand, after it was described by Smith in 1903, it was soon reported from such widely separated areas as Connecticut, Nebraska, Missouri, and Illinois, indicating that it may have been overlooked in other countries by pathologists not familiar with bacterial diseases. That the disease has never been reported on either plum or peach in Europe indicates that it was probably not introduced from that region. There is a possibility that it might have been imported from Japan in recent years since many plum varieties on which the bacteria live for several years in the cankers were imported from Japan as early as 1870 and later by Burbank.

The fact that these were first grown in California where bacterial spot is not yet known would throw doubt on this supposition. A reasonable explanation of its origin is that it was indigenous on certain of the native wild plums in America and from these spread to the peach where peach orchards were planted near the wild plums.

Bacterial spot was first described by Smith in 1902 as a disease on the leaves and fruit of Japanese plums in Michigan. He also established the fact that the infection takes place through the stomata and named the organism *Pseudomonas pruni*. The possible relationship of the peach and plum diseases was soon recognized but not proved until 1908 when Smith successfully inoculated peach with the organism taken from plum. In 1906, Heald found twig and branch cankers on plums in Nebraska which he considered were produced by Smith's *P. pruni*. A monographic study of the disease was published by Rolfs in 1915. He listed additional host plants and recorded his observations on the relative severity of the disease, both leaf and fruit spot, on some 250 varieties of peaches, 60 plum varieties, and some apricot and nectarine varieties growing at the Missouri State Fruit Experiment Station, Mountain Grove, Missouri. Additional host plants were listed by Kuwatsuka (1921), Ishiyama (1922), and Dunegan (1932). Thornberry and Anderson in 1933 reported the discovery of the "spring cankers" and their significance in the overwintering of the bacteria.

Some significant developments in the control of this disease were contributed by Roberts and Pierce (1929) who introduced zinc sulfate–lime, which indirectly proved to be one of the most important spray practices, especially in the prevention of lead arsenate injury. In 1951, Mason reported the successful control of primary infection from the spring cankers by spraying with copper sulfate the preceding fall.

Geographic Distribution. Bacterial spot has been reported from Japan, Australia, Brazil, New Zealand, U.S.S.R., Canada, and the United States. There is also an unconfirmed report from China. In the United States it occurs in all of the peach- and plum-growing states east of the Rocky Mountains. It has not yet been found in the Pacific Coast area.

Economic Importance. As is usually the case with important diseases, the losses sustained from bacterial spot cannot be accurately measured from figures concerning reductions in marketability of fruit alone. The most serious aspect of this disease is usually its devitalization of the tree, with the manifold consequences resulting from defoliation. The reduction in marketability of fruit as the result of direct bacterial invasion or of reduced quantity and quality from defoliation is certainly another serious aspect which may at times appear the most important. It is not uncommon to discard 15 to 30 per cent of the fruit of susceptible varieties in a bad bacterial spot year. Such a figure does not consider the fact that still more of this fruit was infected, but not so badly that it could not be sold in a reduced grade. Direct killing of twigs is occasionally important, but as a rule their death is more a result of general tree devitalization.

Symptoms. The disease occurs on the leaves, twigs, and fruit. The symptoms produced are quite distinguishable from those produced on the peach by other pathogenes. They are, however, easily confused with certain types of spray injury, and when both conditions are present even an experienced observer is likely to require a microscope for an accurate diagnosis. The presence of bacteria in the spots is the most reliable index for separating the two troubles.

On the Leaves. Bacterial spot as it occurs on the leaves of peach is first evident as small circular or somewhat irregular areas, a little lighter in color (pale green to almost white) than the surrounding tissue. The spots are usually small and first visible on the undersurface of the leaf, but soon become evident on the upper surface. As the disease develops, the spots enlarge in size, become angular in outline, and darken to a deep purple, brown, or black (Fig. 48). The color of the tissue immediately surrounding the darkened area often fades to a light yellowish green. Frequently, before there is much change in the color of the spot, a line of cleavage develops around it and when the leaf is bent in the hand, the central area separates entirely from the surrounding tissue and falls out. Normally the central area remains in place for some time after it is separated, then turns to a darker color, and finally falls out. This gives the leaves the characteristic ragged, shot-hole appearance. Quite often the cutting off of the diseased central area checks the spread of the disease from this point, but in many cases another infected ring is formed about the central opening, which in turn is cut off by the surrounding tissue. The mature leaf spots are interveinal, generally angular in outline, and

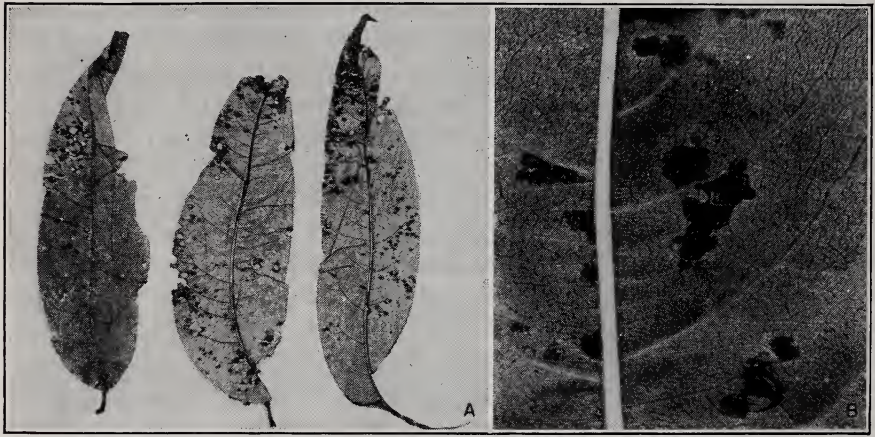

FIG. 48. Bacterial spot on peach leaves. At right, enlarged to show typical angular outline.

from 1 to 5 mm in diameter. While individual spots are small, several may coalesce, involving large areas of the leaf. This grouping of spots is especially common on the tip of the leaf, where often an inch or more may be involved. Badly infected leaves soon turn yellow and drop off; this is especially true on trees of low vigor, or when the disease is supplemented by arsenical injury. On the plum leaves the spots are similar to those in peach but the shot-hole effect is more pronounced.

On the Fruit. Bacterial spot is first evident on the peach fruit as small circular brown spots on the surface. As the disease develops the spots become slightly darker and depressed, and frequently the margins appear "water-soaked." They may appear any place on the fruit surface, but are usually localized. Spots may be few in number or so numerous as to literally cover large areas of the fruit.

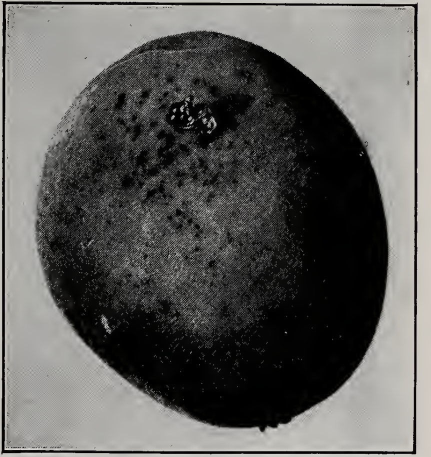

Fig. 49. Bacterial spot on fruit, showing gumming (not common).

The most conspicuous phase of fruit infection is the "pitting" and "cracking" which occurs in the vicinity of fruit spots as a result of the natural growth and enlargement of the fruit (Fig. 49). It is this phase, also, which makes so much of the infected fruit unmarketable. In the event of early severe infection, all imaginable shapes of cracking and fruit deformation may be seen. The most common types encountered are (1) small shallow cracks which do not greatly exceed the area of the original spots, but which may become large and distinct, especially if arsenical sprays without proper "safeners" have been used; (2) the coalescing of many individual areas to form a rather large unsightly lesion; and (3) the "pin-point" type, distinct from any of the ordinary "cracking" types of fruit injury. The cracks which commonly occur are very small, and many times they are scarcely visible to the naked eye. The outstanding feature of this injury is the light green halo which surrounds the spots, especially when they appear on the blush surface of the fruit. This condition, when rather extensive, causes the fruit to appear distinctly mottled but does not greatly influence its market value. Under certain weather conditions a "gum-flow" type of injury occurs. As the name implies, it is characterized by the exudation of gum from the bacterial wound (Fig. 49). It is easily confused with curculio and some other common insect injuries. If the gum is removed, the difference between them is quite evident. This type of injury is usually evident after periods of rainy weather. On *plum* fruit the symptoms may be quite different from those on peach. Large sunken black lesions are common on some varieties, while on others small pitlike lesions are present.

On Twigs. Two distinct types of lesions are recognizable on peach twigs. They have been designated by Thornberry and Anderson as "spring" and "summer" cankers.

The *spring cankers,* developing on young succulent twigs of the past summer's growth, form water-soaked, slightly darkened blisters about the time the first leaves appear. At first they are small raised areas about 2 mm in diameter, but later extend from 1 to 10 cm parallel to the long axis of the twig (Fig. 50). They seldom extend more than one-half the circumference of the twig, but may encircle it, in which case a type of die back results. Later in the spring the epidermis over the lesion is ruptured, and bacteria are exposed for dissemination. The infected area heals over during the growing season. In some seasons, apparently when fall invasion of the bacteria occurs near the terminal bud, a "black tip" type of injury is common. The tip of the twig dies during the winter, and in early spring the living tissue below is rapidly invaded by the bacteria, resulting in a dark area from ¼ to 1 in. in length. After the bacteria are discharged, the entire tip dies back. Terminal die back may result from a number of other causes, in which case a definite line of demarcation between the dead and living tissue may be observed when growth is renewed in early spring.

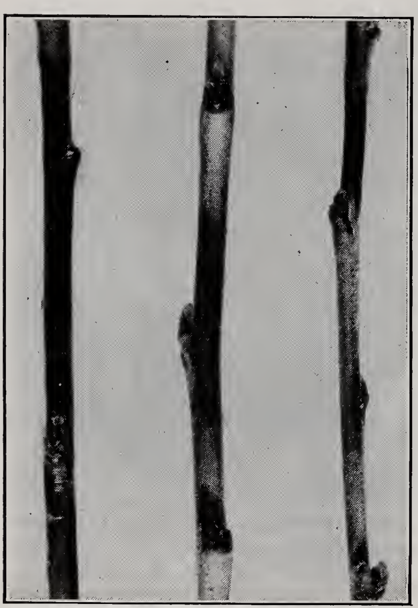

FIG. 50. Bacterial spot spring cankers on peach twigs. Note indefinite dark areas centered at buds.

Summer cankers develop on green shoots later in the summer, usually after foliage infection has become established. At first they are water-soaked, dark purplish spots surrounding stomata, but are not readily recognized until larger twig areas are involved. As the spots develop they appear as brown to purple-black lesions, slightly sunken, circular to elliptical in shape, with a water-soaked margin (Fig. 51). Mature spring and summer cankers vary greatly in size and shape, but generally spring cankers are superficial and extend over a larger area.

The cankers may be quite different on certain plum and apricot varieties. In both of these stone fruits the bacteria survive the winter and continue their development in 2- and 3-year-old twigs. Also penetration of the inner bark occurs. The resulting cankers are deep-seated and often deform the twigs so that they have a knobby appearance. Killing of 2- or 3-year-old twigs often results.

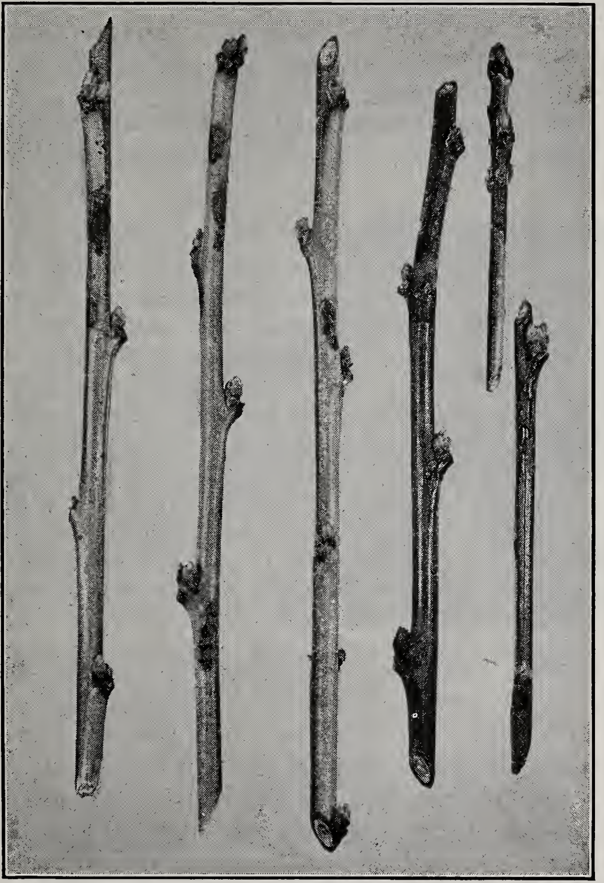

FIG. 51. Bacterial spot summer cankers. Note definite outline of cankers.

Sweet and sour cherries sometimes show leaf infections, but these are rarely serious. The leaf symptoms are much the same as on peach.

Causal Organism.[1] Bacterial spot is caused by a bacterium which was originally described in 1903 as *Pseudomonas pruni* by Smith. Because of the different systems of classification which were employed, the organism has been variously referred to as *Pseudomonas pruni* E. F. Smith, *Bacterium pruni* E. F. Smith, and *Phytomonas pruni* (E. F. Smith) Bergey et al. Since Dowson's reclassification in 1939 *Xanthomonas pruni* (E. F. Smith) Dowson is the accepted name.

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[1] *Xanthomonas pruni* (E.F.S.) Dow. Class Schizomycetes, Order Eubacteriales, Family Pseudomonadaceae.

Hosts. Bacterial spot has been reported only on species of the genus *Prunus.* The common host plants are peach, apricot, nectarine, almond, and plum (Japanese, European, and American species). Sweet and sour cherries are also susceptible. Other host plants listed are Japanese apricot (*P. mume*), Chinese wild peach (*P. davidiana*), *P. buergeriana*, *P. crassipes*, and *P. donarium.* Of the above hosts the most susceptible are peach, apricot, and Japanese plums. American and European plum varieties are less susceptible, but there is a wide varietal difference in all of these groups.

Morphology and Cultural Characters. The organism is a short rod, with rounded ends, which may occur singly or in short chains. It is motile by one to several polar flagella. The bacteria measure from 0.4 to 1.7 μ long and from 0.2 to 0.8 μ wide. Capsules are present in cultures after about 9 days. The organism stains Gram-negative; is not acid fast; is aerobic; is yellow on culture media; slowly liquefies gelatin; gives a slight diastatic action; curdles and peptonizes milk; ferments carbohydrates without the production of gas, but does produce an acid reaction in the media; reduces nitrates; produces indol; grows in Cohn's solution; reduces litmus; has minimum, optimum, and maximum temperatures of 7, 25, and 38°C respectively and a thermal death point of 51 to 52°C. Exposure of the bacteria to the sun's rays inactivates them in from 30 to 45 minutes. They resist the lethal effects of desiccation from 10 to 13 days. Longevity tests indicate that the organism may survive for several months under laboratory and outdoor conditions on culture media.

Disease Cycle. Although *X. pruni* has been demonstrated to survive the winter in fallen leaves, it is doubtful if these bacteria are of much importance in dissemination. Twig cankers are the main source of primary spring infections. In this connection exists one of the most important differences between spring and summer cankers. There is little doubt that in certain localities the organism may survive the winter in a small percentage of summer cankers and produce the bacterial spot disease the following spring. On the other hand, Anderson and Thornberry working under Illinois conditions traced hundreds of summer cankers through the winter, and with only one exception, *X. pruni* was never recovered from them after December first. The tendency of the host tissue to produce a periderm in advance of the invading bacteria, thus quite effectively cutting off the food supply, may have some bearing on the longevity of the organism in these cankers. Dunegan has shown that the bacteria are not viable after 10 to 13 days of desiccation. It is, therefore, possible that the drying out of the cankers, after periderm formation, is responsible for the dying out of the bacteria. Thornberry has shown that *X. pruni* inoculated into young peach twigs at Yonkers, New York, at regular intervals during the dormant season survived the winter and produced typical spring cankers early the following spring. Bacteria thus inoculated are not cut off from healthy tissue by periderm formation. Accordingly, they pass the winter in the presence of plenty of food and moisture, and whenever temperatures are satisfactory for growth the invasion of healthy tissue is resumed.

Spring cankers apparently arise from twig infections in the late summer and fall before leaf fall, but do not become evident until the infected tissue appears discolored and water-soaked the following spring. This fact may explain in part why active cankers are so difficult to locate during the dormant season and yet are plentiful and relatively easy to find about the time the first leaves appear in the spring. The lack of distinction between the two canker types may account for the belief that summer cankers play so important a role in spring dissemination of the bacteria. Many isolations have been made from spring cankers, and in all cases *X. pruni* has been recovered. Thus it would seem that spring cankers are largely responsible for primary infections of *X. pruni.* However, Foster and Petersen in 1954 reported the recovery also of viable bacteria from a few summer cankers in South Carolina collected on Jan. 27, 1953.

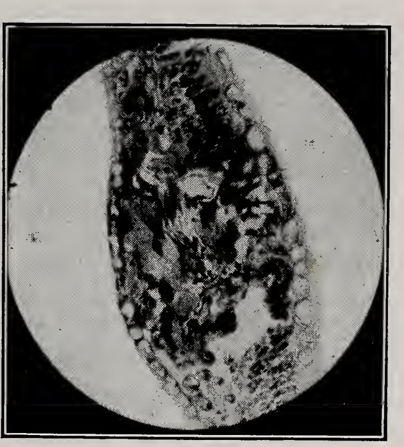

Fig. 52. Bacterial spot. Section of young lesion on leaf, showing masses of bacteria and swelling and disorganization of host cells.

While it seems evident that initial infection in the spring originates from the few spring cankers so far as the peach is concerned, the situation is quite different when we consider the plum and apricot. In these plants the summer cankers formed on the current season's twigs continue developing the following spring, with the bacteria remaining alive throughout the winter. Bacteria have been recovered from 3-year-old plum twigs. This fact is of great importance in the nursery, since peach trees planted near either infected apricot or plum trees are likely to become infected, and when planted in the orchard will produce spring cankers. It is very unusual to find leaf infection in nursery trees where these are separated from blocks of plums or apricots. (See under Control.)

After primary infection of leaves and twigs in the spring (Fig. 52), the extent and severity of the infections which follow depend entirely upon environmental conditions. Infections may occur any time during the growing season on all susceptible parts, i.e., leaves, current-year twigs, and fruit, when moisture and temperature conditions permit. A warm, moderate season with light frequent rains accompanied by fairly high winds and heavy dews, is most favorable for severe infections. In ordinary years the disease appears and spreads during the spring rainy periods, but makes little progress during the hot, dry summer months. Late infections usually accompany fall dews and rains. They are of particular concern on late-maturing varieties because of the damage resulting from fruit infections. Generally speaking, however, the most serious aspect of bacterial spot is the devitalization of infected trees as a result of defoliation. This is especially true if it occurs early and continues throughout the growing season.

Control. The correlation between general tree vigor and control of bacterial spot suggests one of the most important control procedures. A general program of orchard management conducive to the production of vigorous trees should be followed.

Since the bacteria do not live over winter in the fallen leaves nor in visible cankers on the twigs, little if any benefit comes from sanitary measures or from pruning.

It is very seldom that nursery trees are infected when received by the orchardist. Thus, during the first 2 or 3 years in the orchard the trees remain free of the disease unless planted near an older infected orchard. In isolated areas in the absence of neighboring plum or apricot trees, the peach orchard may escape infection for a number of years.

Nurserymen can prevent infection in the nursery by (1) securing budwood from trees free of bacterial spot and (2) planting peach stock in blocks well separated from plum or apricot stock. Nurseries where budwood is taken from older nursery trees rather than from bearing trees seldom show infection.

While varietal differences in susceptibility are marked, there are no good commercial varieties of peaches which are highly resistant. J. H. Hale and hybrids of this variety are all very susceptible.

Many attempts have been made to develop a successful spray for bacterial spot control. Some success has been obtained by the use of zinc sulfate–lime. This spray is of considerable value since it was found to be very effective in the control of arsenical injury when lead arsenate was used as the chief insecticide. It also had some stimulatory effect on orchards where zinc was deficient in the soil.

The application of postharvest sprays for the prevention or eradication of spring cankers has proved highly successful in some years. Experiments carried out in Arkansas and Illinois using copper sulfate at the rate of 4 lb to 100 gal applied about the middle of October almost completely eliminated the spring cankers and consequent primary infection. This treatment failed to give substantial reduction in 1953–1954. Improper timing seemed to be the reason for this failure. Since earlier applications of copper sulfate at this concentration would cause serious injury, a schedule of repeated applications using a lower concentration and adding Triton B1956 was tried. In 1953, three applications of copper sulfate at 1 lb to 100 gal with 3 oz of Triton gave good results without foliage injury. Best results were obtained by applications made on August 16, September 4, and September 18. Since there seems to be no reliable method of determining the period when the bacteria in the twigs are most vulnerable, split applications in the late summer are advised.

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Peach Leaf Curl

The disease now commonly and appropriately known as "peach leaf curl" is also referred to as "curl," "curl leaf," or "leaf blister."

History. Leaf curl has been known in Europe since the early part of the nineteenth century and was among the earliest-recognized peach diseases in the United States. It was recorded from the eastern United States as a well-known malady in 1845 and from California in 1855. In California, according to early horticultural writings, it is suggested that the disease may have been introduced by the Spanish on peach trees from Mexico, since it first appeared on the peach around the missions, and from this source spread to the budded peach trees which were at this time being introduced into California. It was definitely referred to in England as early as 1821, but it was not until 1860 that Berkeley, an English mycologist, accurately described the organism causing the disease. It was recorded from Australia as early as 1856. China has been considered by some as the most likely point of origin of this disease, although there is no conclusive evidence to bear out this contention. It is certain that the disease was well established in European countries by 1880, since scientists of Europe gave prominent consideration to it in this general period.

Although many early contributions were made by European workers with regard to the symptoms and effect of the disease, they were slow in contributing to our knowledge of effective control measures. This may have been due, at least in part, to their rather general and persistent belief that the fungus was perennial in twigs and buds and, accordingly, could be controlled only by the eradication of infected plant parts. In the period 1880 to 1885 California growers began the use of lime, sulfur, and salt for the control of San José scale. They soon observed that if this spray was applied before the buds opened in the spring, leaf curl control also resulted. Experiments at the University of California by Benton in 1890 showed that winter applications of copper sprays were also effective, but that control of leaf curl could not be accomplished after the buds had opened. He further demonstrated that infections from perennial mycelium were the rare exception rather than the rule. In this connection recent work tends to indicate that perennial mycelium of the peach leaf curl fungus does not exist. The copper treatment was independently discovered in Australia in 1891. The successful control of leaf curl was not recorded in Europe until 1898 and in eastern United States in 1899 and 1900. Work on the entire leaf curl problem was begun by Pierce of the U.S. Department of Agriculture in 1893, and in 1900 a monographic treatise was published. Most of our present practices are directly traceable to this excellent study. Mix and Fitzpatrick cleared up many confusing details concerning the life history and infection habits of the peach leaf curl organism. Mix (1935) gave definite proof that the source of infection is from overwintering conidia on the twigs and that these conidia may survive for several years.

Geographic Distribution. Leaf curl practically coexists with the peach, although there are several regions in the world where environmental conditions do not favor the disease. It has been destructive in Europe, parts of China and Japan, the U.S.S.R., Africa, Australia, South America, North America, and New Zealand. The disease is important in the United States in all peach-growing regions except the irrigated sections of Washington and the semiarid regions of the Southwest.

Economic Importance. Considering the ease with which leaf curl may be controlled, it should no longer be one of the most important peach diseases; actually, however, it is still a very serious problem in most regions where peaches are grown. This is due to the fact that an unbelievable number of small growers do not yet know how to combat it. Also, in sections where the disease does not occur regularly, many growers prefer to take the chance that it will not be serious rather than spend a few dollars on spray materials. Before a satisfactory control was known it was estimated that a 3-million-dollar loss of crops occurred annually in the United States alone. Recent estimates by the Plant Disease Survey indicate that some sections of the country still lose annually as much as 15 per cent of the crop.

The loss from leaf curl is caused through defoliation, which, in turn, induces failure of the tree to hold and develop its fruit. Frequently the tree becomes so completely defoliated that new shoots are sent out the same season, thus impairing the vitality of the tree, making it much more subject to winter injury and attacks by other disease-producing fungi. Buds and twigs are also killed, and the final result of successive seasons of injury is the death of the entire tree. The devitalizing effect of leaf curl upon infected trees is undoubtedly as important as the actual loss of crop in any given season.

Symptoms. Leaf curl is usually confined to the current year's growth, involving leaves, blossoms, twigs, and fruit. It rarely extends downward to the growth of the previous season. In the spring when the leaves first appear they are much thickened, and as they develop the leaf blade becomes puffed and folded with the edges curling inward, so that the undersurface of the leaf is a series of concaved chambers (Fig. 53). Most generally the entire leaf shows these symptoms, although it is not uncommon for small portions to be infected. Likewise, only a few leaves on a tree may show the disease, while in other instances infections may be so numerous as practically to destroy the entire foliage. Very shortly after diseased leaves appear, they acquire red or purplish tints, making them especially conspicuous. Chlorophyll soon completely disappears, and the bright colors fade into a reddish yellow or yellowish gray. Usually at this stage the fungus begins to produce spores on the upper surface of the leaves. This fruiting of the fungus causes the leaves to acquire a powdery-gray coating. Soon after this condition is reached the leaves turn a yellowish brown to brown, wither, and fall off. The actual time of leaf fall depends largely upon weather conditions which prevail. Dry hot weather hastens leaf drop, which usually occurs in late June or early July. If any considerable portion of the leaves is dropped it will usually be replaced by leaves from dormant buds.

On twigs the disease causes a swelling, which is noticeable only in cases of severe infection. The twigs may have a slightly yellowish cast and, as a rule, are decidedly stunted. Infected blossoms and fruit practically always fall early in the season, although it is not uncommon to find some diseased fruit at harvest. On such fruit the lesions are usually local and evident as shiny, raised, warty areas. The color pattern so typical of leaf infections is also present on infected fruit, although not generally so pronounced.

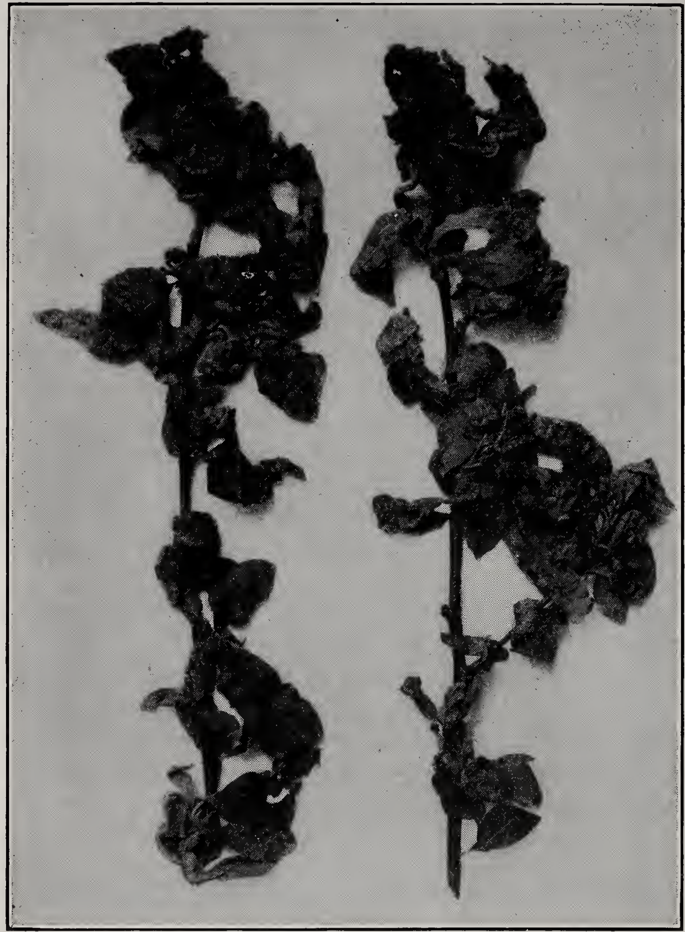

Fig. 53. Peach leaf curl.

Causal Organism. The fungus causing peach leaf curl was first described by Berkeley in 1857 as *Ascomyces deformans*, but the name was changed in 1869 by Fuckel to *Exoascus deformans*. In 1866 Tulasne described the organism as *Taphrina deformans*,[1] which is the binomial generally employed by current writers.

Hosts. Leaf curl caused by *T. deformans* (Berk.) Tul. is principally a disease of the peach. Certain peach relatives, such as the nectarine and almond, are reported as susceptible, while the apricot is immune. There are some varietal differences, but they are not marked. Seedlings are usually more susceptible than the standard commercial varieties.

Morphology. The *mycelium* of peach leaf curl has been shown to assume some very different and distinct hyphal types. Pierce has recognized vegetative, distributive, and ascogenous forms which are probably distinguishable, for the most part, as a physiological response to the availability of food or the ease of penetration as found in different types of tissue. The hypha which he has designated as vegetative is composed of relatively short, irregularly shaped cells, which vary greatly in size and are usually curved and twisted. This type of growth is most generally found in parenchyma tissue and is intercellular.

Fruiting or ascogenous hyphae have been observed to arise from well-developed vegetative mycelium. Large, well-developed vegetative hyphae commonly form just below the epidermis of the upper and occasionally the lower surface of the leaf. They penetrate between the cells of the epidermis and grow freely in all directions between the epidermis and the cuticle. The hymenial layer is soon in evidence as aggregates of mycelium practically covering the epidermis of the leaf beneath the cuticle. As the hymenium matures, the cells begin to distend and, shortened by new transverse septa, continue for some time to enlarge. This entire process continues until the cells of the hymenium become quite uniform in size and shape and form a continuous mat over large areas of infected tissue. At this stage the hymenium is fully developed and ready to produce ascospores.

Asci originate from ascogenous cells as thin-walled protuberances which may penetrate or, in the case of large numbers, actually lift and break the cuticle. When such outgrowths are numerous and produce ascospores and bud conidia, the typical gray, plushlike, or powdery surface growth is evident. The contents of a forming ascus is finely granular, and as the ascus elongates it crowds into the upper portion, which is soon cut off from the parent ascogenous cell by the formation of a septum somewhere near the base of the ascus. This basal cell is called the "stalk cell." A mature ascus is typically clavate, being broader at the top than at the base. Asci usually vary greatly in size from 17 to 36 μ long and from 7 to 15 μ wide. Eight round or oval *ascospores* are usually formed. They vary in size from 3 to 7 μ in diameter. The ascospores escape from the ascus through an apical rupture.

Ascospores commonly germinate by budding (formation of *bud conidia*) and by the production of stocky germ tubes. Budding may occur before or after the ascospores escape from the ascus. When bud conidia are formed the process may take place from the ascospore directly, one conidium after another being produced, or the contents of the ascospore may pass into a thin-walled conidium of about equal size; this conidium then assumes the function of bud production. Ascospore budding usually occurs at one end of the spore, although occasionally buds arise laterally. Under favorable conditions, ascospores will produce conidia continually until the entire ascospore content is depleted. The budding of newly formed bud conidia is also of common occurrence and is the common vegetative type of growth and multiplication.

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[1] *Taphrina deformans* (Berk.) Tul., Class Ascomycetes, Order Protodiscales, Family Exoascaceae.

Recent workers contend that conidia may survive on all the surfaces of the dormant tree. They have been shown to remain viable under field conditions for more than one year and under laboratory conditions as long as eleven years.

Disease Cycle. The ascospores of *T. deformans* or the conidia produced from them carry the fungus through the summer, fall, and winter months lodged on the surface of trees or on the ground beneath them. With the advent of spring and the swelling of the buds, if conditions of moisture and temperature are suitable, the spores germinate, and those which come in contact with the young opening leaves cause infections. The infection spore fastens itself to the undersurface of the young leaf by the production of a short peglike germ tube which penetrates the epidermal wall. Once penetration of the epidermis is accomplished, the fungus spreads out in the tissues between the lower and upper epidermal walls. The typical swelling of infected tissue is soon evident. The asci soon develop and discharge their spores immediately. Thus it may be seen that from the time of initial spring infection until ascospores and bud conidia are formed and discharged is only a matter of a few weeks. After spore discharge the fungus dies out and the leaf falls unless only a very small area was infected. There is no evidence that the fungus becomes perennial in infected twigs, as was thought by so many early workers. It is supposed that the ascospores and bud conidia lodged on the tree surface produce great numbers of secondary conidia whenever suitable weather conditions prevail. During hot dry periods of summer and the cold of winter the conidia form thick-walled resting cells. This saprophytic development of conidia is held to account for severe outbreaks of leaf curl following years of very light infections. There is good evidence that conidia may remain viable on the twigs for 2 or more years. Thus the fact that leaf curl was absent or very light the preceding spring is no assurance that the disease will not develop the following spring if weather conditions are favorable. While secondary infections occur under very favorable conditions, they are of little economic importance. From this account of the life cycle it may be noted that after infected leaves fall, the fungus is reestablished on and beneath the tree ready to infect the opening leaves the following spring.

Relation to environment is one of the most important considerations concerning the general behavior of peach leaf curl. In the few parts of the world where leaf curl is either seldom serious or never appears, environment is thought to be the limiting factor of its development. It is suggested that the dry hot summers so common in southwestern United States are too extreme for leaf curl spores. In this connection it has been demonstrated that the upper limits of viability of peach leaf curl in culture are from 79 to 86°F. The optimum temperature for its development is about 68°F, and the minimum is below 40°F. It is not unlikely that prolonged exposures of the spores to temperatures above 86°F would materially reduce their viability or actually kill them. There are other sections of the world where moisture is more likely the limiting factor. The actual period of infection has been determined to be a relatively short time during the swelling and opening of leaf buds in the spring. Rains during this period are necessary to produce infections. The exact relation of rains to infections, aside from the necessity of moisture for spore germination, is still problematical. It has been suggested that heavy rains cause the host tissue to become succulent and distended with water and consequently relatively more subject to invasion. Prolonged periods of cold rainy weather in the spring retard tree growth more in proportion than fungus growth, thus extending the infection period. Whatever the relationship, it is well established that inoculum being present, cold wet weather when the buds are swelling and opening is conducive to epiphytotics. Regions of light dews, light rainfall, and early warm spring weather are generally unfavorable for the disease.

Control. Leaf curl is one of the relatively few serious diseases of peach which may be cheaply and effectively controlled. If sprays are applied at the correct time and the correct materials used, a 95 to 98 per cent control is usually accomplished. It has been repeatedly demonstrated that leaf curl may be controlled by *one* application of a suitable fungicide during the dormant season. The spray is most effective when applied just before the swelling of the buds, but in actual commercial practice 3 to 4 weeks should be allowed as a margin of safety. It should be emphasized that leaf curl cannot be controlled after the disease has entered the host tissue in the spring and that applications *must* be made before the buds begin to swell.

It has been observed, under Illinois conditions, that sulfur dusts or sprays applied for the control of peach brown rot just prior to harvest controls leaf curl the following spring.

The possibility of the selection of resistant varieties has been given very little attention because of the satisfactory leaf curl control by chemicals. Variation in severity of infection of varieties to the leaf curl disease has been observed.

Several hundred varieties of peach and nectarine of foreign and domestic origin were surveyed at Chico, California, following a severe and general outbreak of leaf curl. Only a few of the foreign importations were free of the disease. Thus, there seems to be little chance that any variety is resistant enough to warrant selection by the grower.

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Plum Pockets

A number of diseases of species of *Prunus* are caused by fungi similar to *Taphrina deformans* (see page 226). Of these diseases "plum pockets" is the most important from a commercial standpoint. It is also known by the common names "bladder plums," "mock plums," and "fools."

History. The problem of determining the original home of the fungi causing plum pockets is complicated by the fact that the two species commonly causing the disease were confused by early writers. It now seems reasonably certain that the American species (*T. communis*) is confined to American species of plums, while the common European species (*T. pruni*) is rarely found in North America but is very common in a number of European species of *Prunus*. Thus it seems likely that the American species was present on wild plums before this country was settled and that the European species originated in the native home of the European plums. No reference has been found in early horticultural writings to this disease.

T. pruni was described in the middle of the nineteenth century, while the American species was not described until 1893, although the disease was recognized and described by botanists a number of years earlier.

Geographic Distribution. Plum pockets caused by the various species of *Taphrina* is world-wide in distribution, although the causal fungus species are somewhat restricted in their distribution. For example, *T. communis* is found mostly in eastern and middle North America, while *T. pruni* is limited to Europe and Japan. Another American species, *T. pruni-subcordata,* is found only in the Rocky Mountain and Pacific Coast area on *Prunus subcordata.*

Economic Importance. Losses from plum pockets may be extremely heavy in some seasons. For example, in northern United States where only the American varieties of plums are grown, 50 per cent or more of the fruit may be lost in years when the disease is severe. In addition, serious shoot injury occurs since the fungus causes hypertrophy and final death of the young shoots. In Europe, the European species attacks all of the common European plum species and may cause extensive damage in a year favorable to the disease. From a commercial standpoint the total loss from this disease is not great.

Symptoms. The first evidence of the disease on the fruit is the appearance of small whitish spots, or blisters, about 2 or 3 weeks after shuck fall. These blisters enlarge rapidly as the fruit develops, and soon the entire fruit is involved. The flesh becomes spongy, and the young seed ceases development, turns brown, and withers, leaving a hollow cavity. The fruit enlarges rapidly, so that it is soon ten or more times the size of the healthy plums (Fig. 54). It is so misshaped that it is hardly recognizable as a plum. At first reddish, it later has a velvety gray appearance. The spongy interior dries up, leaving only an exterior shell. This hollow distorted structure is the characteristic which gave rise to the common name "bladder plum."

The developing shoots and leaves are often infected. The tips of the shoots become greatly enlarged and are often twisted or curled. The leaves are deformed and curled as in leaf curl of peach, but are not so conspicuous.

Causal Organism.[1] Mix, in his monograph of the genus *Taphrina,* has done much to clear up the synonomy of the two species causing plum pockets on commercial fruit crops. He has combined several species, mainly on the basis of morphological or cultural characters. A more extensive study of host reactions by cross inoculation might throw additional light on the presence of biologic forms or the separation of species, but the classification as given by Mix is accepted as logical. *T. pruni* was described by Tulasne in 1866. Synonyms under the genus

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[1] *Taphrina* spp. Class Ascomycetes, Order Protodiscales, Family Exoascacae.

Exoascus are *E. rostrupianus* Sadeb., 1893, and *E. insititiae* Sadeb., 1884. *T. communis* was described under the genus *Exoascus* in 1893 by Sadebeck. Two years later it was transferred to *Taphrina* by Giesenhagen. Synonyms as given by Mix are *E. longpipes* Atk. and *E. mirabilis* Atk. All of these were transferred to *Taphrina* by Giesenhagen in his revision of the genus.

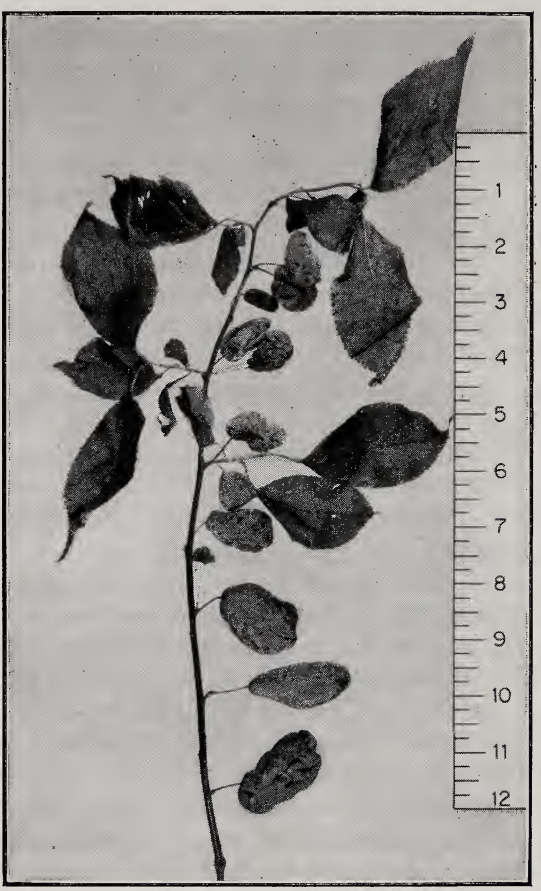

Fig. 54. Plum pockets on fruit.

Hosts. *T. communis* has been found mainly on American plums including: wild goose or red plum (*P. americana*), chickasaw (*P. augustifolia*),[1] wild goose (*P. hortulana* and *P. munsoniana*), beach plum (*P. maritima*), Canada plum (*P. nigra*), *P. lantana*, *P. salicina*, and *P. umbelata*.

[1] The classification of *Prunus* here used is mainly based on that of Rehder in his Manual of cultivated trees and shrubs, 2d ed., The Macmillan Company, New York, 1940.

T. pruni, found mainly on European plums, has been reported on the common garden plum (*P. domestica*), cherry plum (*P. cerasifera* var. *divericata*), Damson (*P. insititia*) Sloe, or Blackthorn (*P. spinosa*), and *P. ussuriensis.*

Morphology. The *mycelium* is much like that described under peach leaf curl. For the most part it develops in the intercellular spaces, later forming a subcuticular layer of isodiometric cells. The *asci* are formed from a layer of subcuticular ascogenous cells with no ascocarp developing. The asci of both species are cylindric-clavate, rounded or truncate at the apex. Dimensions are for *T. pruni* 17 to 53 by 5 to 17 μ and for *T. communis* 27 to 83 by 5 to 13 μ. *Ascospores,* round to ovate, commonly budding in the ascus are 4 to 7 by 3 to 6 μ for *T. pruni* and 4 to 7 by 3.5 to 5.5 μ for *T. communis.* A notable characteristic of *T. communis* is the presence of occasional long asci which project well above the general surface of the hymenium. The *conidia* (*blastospores*) and vegetative growth by budding is as in *T. deformans.*

Disease Cycle. The disease cycle of the plum pockets fungi is much the same as for peach leaf curl, although there is no positive evidence as to the origin of spring infection. It is probable that this arises from conidia (blastospores) which have persisted throughout the summer and winter on the twigs and buds. It is known that infection occurs shortly after the flower buds open. Fruit infection is much more common than is the case with peach leaf curl, and the hypertrophy of fruit and shoots is much more pronounced. Penetration of the hyphae to the seed is very rapid following infection. The hymenial layer of asci is much more common on the fruit and shoots than on the leaves. The discharge of ascospores and their budding gives the surface a gray velvety appearance. Later this turns black as the spongy flesh dries up and forms the hollow "bladder."

Relation to Environment. Cool, wet conditions during the early blooming period are essential for the initiation of the disease.

Control. In North America, it is fortunate that most commercial plums are of European or Asiatic origin and in the absence of the European species of *Taphrina* are not subject to the disease. Little is known about the resistance of hybrids derived from crosses between the European and American species or about varietal susceptibility.

Spraying has been found effective in control when applied early in the spring before the flower buds open. Bordeaux mixture (8-4-100) may be used in cases where the disease is troublesome.

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See further references under Leaf Curl.

Other Stone Fruit Diseases Caused by Taphrina Species

Seventeen species of *Taphrina* are listed by Mix as parasitic on stone fruits. In addition to the peach leaf curl and plum pocket species there are 14 others which are of minor importance, occurring as they do mostly on wild or ornamental host plants. Of these, two occur on apricot, eleven on cherries (mostly wild), and one on a western wild plum. While most of these species attack stone fruits of little economic importance, they are listed below in order to help the reader understand the relation of these species to those commonly met with on cultivated fruits.

T. cerasi causes leaf curl and witches'-broom on cultivated cherries (*Prunus avium* and *P. cerasus*) and a number of wild species. This is the most important of the *Taphrina*-induced diseases of cherries and is commonly called "cherry leaf curl." It is world-wide in its distribution, having been reported from Europe, North America, Australia, New Zealand, South Africa, and Japan. Although it attacks cultivated cherries, it has never been considered of commercial importance.

T. mume causes a leaf curl and hypertrophy of twigs of apricot (*Prunus armeniaca* var. *ansu*), *P. mandshurica*, and *P. mume* in Japan and China.

T. armeniacae causes witches'-broom on apricot. At present it is known to occur only in Rumania.

T. mexicana causes small witches'-broom on a Mexican cherry *P. microphylla*, reported only from Mexico.

T. cerasi-microcarpae causes "pockets" of fruit of Asiatic cherries (*P. humilis*, *P. microcarpa*, and *P. tomentosa*).

T. flavorubra causes smallish elongated pointed "plum pockets" and thickened deformed shoots on western sand cherry (*P. besseyi*) and sand cherry (*P. pumila*) in North America.

T. trunicola causes malformation of shoots of two Japanese cherries in Japan.

T. flectans causes leaf curl and witches'-broom of a western wild cherry (*P. emarginata*) in the Pacific Coast region of North America.

T. farlowii causes leaf curl, thickened malformed twigs, and fruit "pockets" on wild black cherry (*P. serotina*) in eastern North America.

T. andina causes leaf curl, witches'-broom, and greatly elongated fruit deformations (pockets) on a Central and South American wild cherry (*P. serotina* var. *salicifolia*) in Mexico and Ecuador.

T. padi causes "pockets" on the European bird cherry (*P. padus*) in Europe.

T. confusa causes deformed floral parts and fruits of chokecherry (*P.

virginiana and P. alabamensis). It is widely distributed throughout North America where the hosts occur.

T. thomasii causes witches'-broom of cherry laurel (*P. ilicifolia*) in California.

T. pruni-subcordatae, a species closely related to *T. communis,* occurs on the Pacific plum (*P. subcordata*) in the Rocky Mountains and Pacific Coast area of North America. It causes "pockets" and malformed shoots much as described for *T. communis* and *T. pruni.*

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Peach Scab

History. Peach scab, also called "freckles," or "black spot," was no doubt known to peach growers long before it was definitely described by botanists. According to older orchardists in Illinois, for example, "freckles" was regarded as a natural phenomenon on some of the susceptible varieties, caused, as they claimed, by the sun on the exposed face of the peach. The first definite record is a collection by Earle in 1881 from Cobden, Illinois. This fruit specimen was deposited in the herbarium of the New York Botanical Garden.

The fungus causing peach scab was described by von Thümen in 1877 from fruit specimens collected in Austria and was named *Cladosporium carpophilum,* the name now used. The presence of the fungus on leaves and twigs was observed and described later.

There is no evidence as to the origin of the disease, but since the fungus lives over on peach twigs, it was probably introduced into America from Europe when the first living trees were brought over.

Geographic Distribution. Peach scab is known to occur wherever the crop is grown and where weather conditions are favorable for its development.

Economic Importance. The loss from peach scab is great in seasons and areas favorable for its development. The general use of fungicides by commercial growers for brown rot control has reduced the loss from scab to the point where it is of minor importance in most peach-growing areas. The main loss where the disease occurs is from the unsightly marking of the fruit, thus reducing its sale value. In addition, the fruit is often cracked and later rotted by invasion of the brown rot or *Rhizopus* rot fungi. *Leaf infections* often result in serious defoliation, but only where the disease is unusually prevalent. Seedlings in the nursery rows may be dwarfed by the attack of the fungus. *Twig* infections because of their superficial character cause very little damage.

Symptoms. The character of the disease varies with the severity of infection.

On the fruit, when first observable, the diseased areas are small, poorly defined, olivaceous spots less than 1/16 in. in diameter, usually appearing on the upper or exposed surface. When these spots have developed for a few days they appear as distinct, circular, dark-olivaceous lesions, usually from 1/16 to 1/8 in. in diameter. They may be numerous and almost in contact on the upper face of the fruit, more scattered on the sides, and entirely absent on the protected lower face. When infection is severe, these individual spots merge, forming a uniform, dark-olivaceous, velvety blotch over the entire exposed surface and frequently covering one-half of the fruit (Fig. 55). The blotch is superficial, never penetrating below the epidermal layer of cells, but the effect on these cells prevents normal development and results in misshapen fruit. An extra thick, corky layer of cells is produced below the surface of the scabbed area, forming an undesirable tough skin. The inability of this skin to expand with the rapid growth of the peach causes the abnormal shape and cracking.

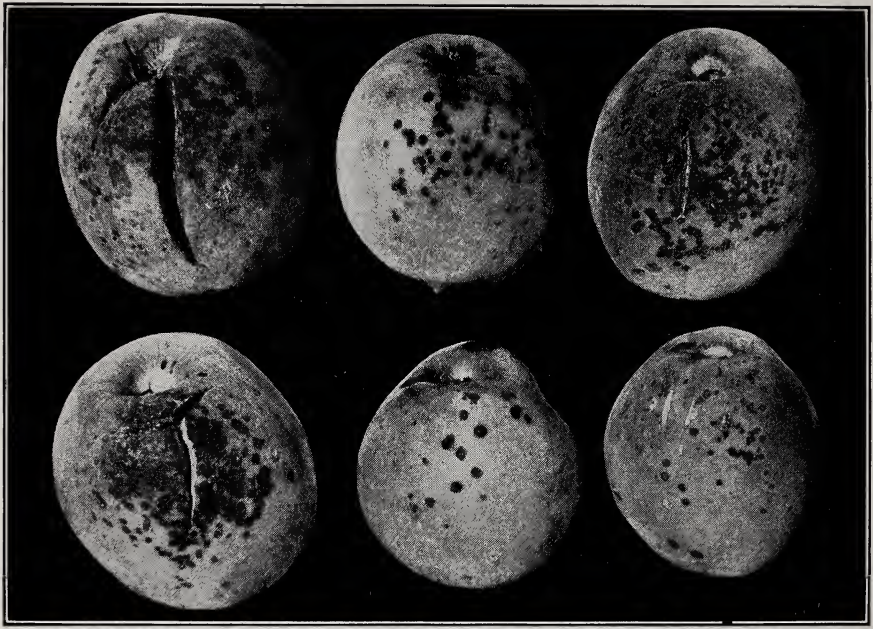

Fig. 55. Peach scab on fruit, showing different types of spotting.

On the twigs, infection is indicated by light brown, oval lesions about 1/8 by 1/4 in. in diameter, which later enlarge and become dark brown. The diseased area is slightly raised, and at the edge there is a peripheral ridge of almost normal color. The injury on the twigs, as on the fruit, is entirely superficial. In the spring olivaceous, velvety dots appear on the surface of these lesions. These twig lesions are first evident on the young tender twigs of the current year at about the time the spots appear on the fruit. They are usually not plainly evident, however, until later in the season. In some cases they are so numerous as to coalesce, giving rise to large, irregular-shaped areas covering the entire surface of the twig for some distance. While the disease on the twigs is of great importance from the standpoint of the wintering of the fungus, the direct injury is not great on account of the superficial character of the growth.

On the leaves, the lesions first appear on the undersurfaces as ill-defined, angular, pale green areas, which later become dark green. The individual spots are usually quite small, rarely developing beyond ¼ in. in diameter. On the midrib, long narrow lesions are developed, usually dark brown in color.

Causal Organism.[1] The fungus causing peach scab, *Cladosporium carpophilum*, belongs in the Fungi Imperfecti and in many respects resembles the apple scab fungus.

Hosts. The peach scab fungus has been reported as occurring on a large number of stone fruit species, including plums (American and European), cherries (sweet and sour), and apricots. Cross inoculations by Bensaude and Keitt in 1921 to 1922 indicated that the *Cladosporiums* fall into two groups: (1) those from peach and plum (*P. americana*) and (2) that from cherry (*P. cerasus*). The peach and plum form infected apricots readily, but not cherry or the European plum (*P. domestica*). They tentatively referred the cherry fungus to *Venturia cerasi* Aderh. [synonym, *Cladosporium cerasi* (Rbh) Sacc.], since it differed in other respects from the peach scab fungus and did not infect peach, apricot, or American plum.

Morphology. The *mycelium* varies greatly under different conditions. The young hyphae are hyaline, septate, and branched. Later the cells thicken and become olivaceous, finally forming a pseudoparenchymatous layer from which the conidiophores arise. The *conidiophores* are usually unbranched, one- to several-septate, and vary greatly in length, but are usually about 30 to 40 μ at the time the first spores are cut off. The *conidia* are borne either singly or in short chains at the end of the conidiophores. They are ordinarily one-celled, but occasionally two-celled, in which case there is a slight constriction near the middle. The conidia measure 14 to 18 by 4 to 5 μ, are fusoid-ovate in shape, and almost hyaline, but with a slight olive-green color.

Disease Cycle. Primary infection arises from conidia produced in the spring on the twig lesions formed the previous year. The conidia remain firmly attached to the conidiophores until moistened; thus dispersal is mostly by washing or by wind-blown mist. On the fruit, infection usually does not occur until several weeks after shuck fall. This is probably due in part to the fact that the hairy condition of the young fruit prevents the spores from coming in contact with the fruit surface. Infection on the leaves is usually on the undersurface.

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[1] *Cladosporium carpophilum* Thüm, Class Fungi Imperfecti, Order Moniliales, Family Dematiaceae.

On germination, the germ tube grows for some distance before penetrating the cuticle. On the fruit the young hyphae may develop for a time around the base of the hairs before infection takes place. After penetrating the cuticle, the hyphae develop in strands or mats, between the cuticle and the outer wall of the epidermal cells. From these the conidiophores push up through the cuticle to form a dark gray to almost black fruiting layer.

The time period between inoculation and the first appearance of the disease (or incubation period) is remarkably long, in the case of the fruit from 40 to 70 days; thus while the spots do not become conspicuous until the fruit is well grown, inoculation may have occurred shortly after shuck fall. The incubation period on the leaves and twigs is from 25 to 45 days.

Spores on the fruit may reinfect the fruit, but since they are produced so late in the season, may not show lesions, except on very late varieties. The twigs of the current season are infected and develop the lesions during the latter part of the summer. While conidia may be produced later in the season, they are of no importance in the life cycle, but these lesions the following spring are the main source of primary infection. Conidia, usually few in number, are also produced on the leaves, but here again they are formed late in the season. Since the twig lesions continue to produce conidia over a fairly long period, secondary inoculation and infection may occur throughout the growing season. This is of special importance in the continuous production of twig lesions. When kept dry the conidia are viable over a period of 3 months or more. This fact is not important, since as a rule abundant freshly produced conidia are present. There is no evidence that the conidia live over winter or that they are produced on fallen leaves or fruit.

No perfect stage of this fungus has been found in spite of many efforts to discover it. If found it would probably be similar to *Venturia inaequalis*, but since the imperfect stage on the twigs furnishes abundant spores for the initial infection, the perfect stage obviously would be of little importance in the life cycle.

Relation to Environment. The optimum temperature for germination and growth is between 65 and 75°F. Spore germination may occur around 37°F and at temperatures as high as 95°F. Thus the usual spring temperatures which prevail after shuck fall are ideal for the development of the fungus.

Moist conditions are necessary for the liberation and dispersal of the spores. Because of the long incubation period, growers are often puzzled by the fact that scab may appear during comparatively dry summers, forgetting the wet conditions which probably existed during the incubation period.

Scab usually occurs in epidemic form only under conditions of moist weather during spring and early summer and on low ground or shaded areas in the orchard.

Control. Considerable difference exists as to varietal susceptibility, but most commercial varieties are fairly susceptible. Since the disease can be readily controlled by spraying, little attention need be given to the selection of resistant varieties for this disease alone.

Cultural practices are very important in preventing infection. Low-lying planting sites should be avoided, and the trees should be thinned by pruning to allow free air circulation.

Most of the standard fungicide sprays are effective in control provided they are properly timed. The most important applications are those made from 2 to 4 weeks after shuck fall. Since insecticides must be applied at this time, the practice of including a fungicide in the curculio sprays should be followed. Sprays for brown rot control later in the season will control any late scab infections. Spraying also prevents the development of twig infections and thus reduces the chance of primary infection the following year.

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Black Knot of Plum and Cherry

Black knot is aptly named, since the conspicuous coal black knotty excrescences on the branches are its most characteristic symptom.

History. The disease was first described by Schweinitz in 1821 from specimens collected in Pennsylvania. Berkeley, an English mycologist, stated in 1856 that black knot was not known to occur in Europe. It seemed to be confined to the eastern United States until around 1875, after which it was reported from several localities in the central states.

Geographic Distribution. Black knot on cultivated plums or wild cherries is generally distributed over North America. It is generally worse in the north sections of the country. It has been reported as occurring in New Zealand.

Economic Importance. Trees with black knot become almost worthless after a few years. Limbs may be killed by the girdling action of the fungus, but the main effect is in stunting and dwarfing resulting from the attack. In addition, the old knots become riddled with insect borings. Probably the greatest damage occurs in small home orchards where no control measures are attempted.

Symptoms. The elongated swellings on the smaller limbs are usually the first indication of the disease observed by the grower. These knots are usually from ½ to 6 or 8 in. in length and from ¼ to 1½ in. in diameter. They ordinarily appear on only one side of the limb, but may encircle the branch. As a rule they are more abundant on the smaller twigs, but as the disease becomes more severe, they may appear on many of the larger branches, especially near the points where small twigs have been produced. The knots are greenish in color and quite soft in texture when first formed, but later become coal black and hard (Fig. 56). The old knots often harbor insects which bring about the death of the twigs if the disease does not.

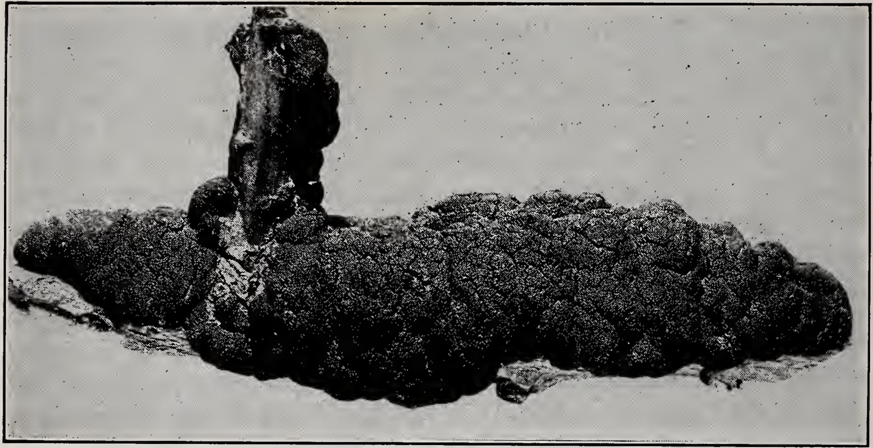

FIG. 56. Black knot of plum, showing minute elevations below which perithecia are developing (× 3).

Frequently a pink mold is observed covering the knots during the summer or fall. This is not a stage in the life of the black knot organism, but is *Cephalothecium roseum*, the fungus which frequently follows scab on apples.

Causal Organism. The ascomycetous fungus causing black knot is *Dibotryon morbosum* (Sch.) Theissen and Sydow.[1] It was first named *Sphaeria morbosa* by Schweinitz in 1821. Saccardo in 1883 assigned it to the order Dothidiales and called it *Plowrightia morbosa*, a name generally used until recently. It has at various times been placed in the genera *Gibbera*, *Cucurbitaria*, *Othiella*, and *Botryosphaeria*.

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[1] Class Ascomycetes, Order Dothidiales, Family Dothidiaceae.

Hosts. The black knot fungus attacks many species of wild and cultivated plums and cherries but has never been reported on peach. It occurs on American, European, and Japanese varieties of cultivated plums and prunes and on both sweet and sour cherries. The problem of varietal susceptibility is complicated by the fact that in some localities an apparently resistant variety shows little resistance in another locality. From this it is deduced that biologic forms exist, but this has not been proved by cross inoculation.

Morphology. The *perithecia* are produced on the surface of black knots as hemispherical, closely appressed elevations. The clavate *asci* are about 120 μ long. The *ascospores* are two-celled (with one cell much smaller than the other), ovate, light green or hyaline, 16 to 12 by 8 to 10 μ. The *conidia* produced early in the spring on the greenish elevations formed the previous year arise from the apex of flexuose, septate, brown conidiophores. The conidia are ovate, light brown in color, and vary in size from 3 to 20 by 2 to 5.5 μ. The conidial stage falls in the genus *Hormodendrum* of the Fungi Imperfecti. *Chlamydospore* formation occurs under certain conditions.

The hypertrophied host tissue is often interspersed with the mycelium of the fungus, but is composed mostly of disorganized host tissue.

Disease Cycle. Primary infection occurs mainly on the wood of the current season's growth, either from ascospores or conidia during a relatively short period around the time of full bloom and extending to beyond the shuck fall stage. The conidia are produced in abundance at this time on the greenish swellings developed the previous year. In most cases the ascospores are discharged from 2-year-old knots, but in some cases perithecia are produced on infections of the previous spring. Penetration may take place through the unwounded surface. The hyphae grow inward to the cambial layer and later along the medullary rays in the xylem portion of the twig. It appears that for 5 or 6 months after infection there is no actual killing of the host cells by the fungus. After invasion of the cambial region the formation of excessive quantities of parenchyma cells occurs, which are pushed outward, thus forming the basis for the knots. The swelling of the tissue becomes evident toward the end of the growing season, and in some cases a crop of conidia is produced in the fall, but usually further development does not occur until the following spring. During the fall and winter the mycelium may spread internally and give rise to secondary galls at some distance from the original knot. During the second summer the knots enlarge very rapidly and the fungus develops the stromatic surface layer in which the perithecia develop during the winter. Ascospores are discharged in the spring and may be borne by air currents or washing to the developing shoots. Often in the second summer these second-year knots are covered with the growth of a pink fungus (*Cephalo-*

thecium roseum), which according to Koch is parasitic on the black knot fungus.

Control. All small shoots and branches showing the swellings should be cut off and burned during the winter or before April 1. Knots on older limbs which need to be saved can be removed by cutting out the diseased area down to the wood and for a distance of 2 or 3 in. above and below the swellings.

Spraying has been effective in control. A delayed dormant of liquid lime sulfur (1:8) followed by a full bloom spray (1:50) and a shuck spray (1:50) will give satisfactory control. It is probable that the full-bloom spray is the most effective, since it is at this time that inoculation occurs.

Removal of wild plum and cherry trees in the neighborhood of the orchard is advisable, especially if these show any signs of the disease.

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Cherry Leaf Spot

This most serious disease of cherries is also known as "yellow leaf," since the bright yellow foliage developing soon after the leaf spots appear is a conspicuous symptom of the disease, especially on the sour cherry varieties. It has also been referred to as "shot-hole disease," since the dead areas of the circular spots often drop out.

History. The first description of the fungus causing leaf spot was by Karsten (1885) in Finland on the European bird cherry (*Prunus padus*). Aderhold reported the disease as having been common on sweet and sour cherries in Europe ten years prior to 1901. In America it was described by Peck from New York in 1878 under the name of *Septoria cerasina* from wild black cherry (*P. serotina*). While the disease was probably prevalent and widespread previous to this time, no record has been found in early horticultural literature of the disease in cultivated cherries. Arthur observed the disease on plums in New York in 1886 and described the perfect stage, but did not name it. Whether this disease is of European or American origin is complicated by the fact that closely related species, or possibly "forms," occur on plums and species of wild cherries. These were not separated into distinct species until Higgins in 1914 and Keitt in 1918 reported extensive morphological and cross-inoculation studies. That cherry leaf spot was generally distributed over the eastern half of the United States was indicated by Pammel in 1891, who stated that it was so destructive in Iowa nurseries that cherries could not be grown from the pits.

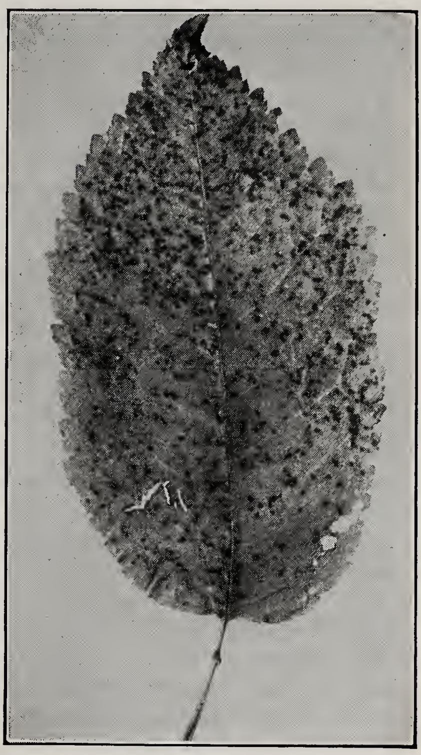

Fig. 57. Cherry leaf spot, upper surface.

Geographic Distribution. Cherry leaf spot is generally distributed and destructive over the United States and Canada where humid conditions prevail. It has also been reported from most European countries on both plums and cherries in recent years, as well as from Australia and Japan.

Economic Importance. Early defoliation causes dwarfed, unevenly ripened fruit with insipid taste. The most serious effect occurs in the season after extensive defoliation. This may result in (1) death of trees, possibly from winter injury, (2) smaller fruit buds and flowers, (3) death of spurs during winter, (4) reduction in set of fruit and fruit size, and (5) reduced growth of shoots. Some of these effects may be noticeable for two or three seasons following defoliation. The over-all effect is a decided reduction of yield, in addition to loss of some trees.

Symptoms. The disease lesions may be formed on leaves, petioles, fruit, and pedicels. During the latter part of May and the first part of June, minute purple spots appear on the upper surfaces of the leaves, especially on any young seedlings or basal sprouts that may be present in the orchard or upon the young trees in the nursery rows. These spots enlarge slightly, but remain quite small when the infection is heavy and the spots numerous (Fig. 57). The outline of the lesion is usually not well defined in its early stages. Later the area in the center of the spot may die and fall out, giving a shot-hole effect.

On the undersurfaces of the leaves following heavy dews or rains, there may be observed a white mildewlike growth. Many of the leaves turn yellow before falling, thus giving rise to the name "yellow leaf." Frequently there is an area about the spots which remains green, and this gives the leaf a mottled appearance. The leaves fall off early in the season, and thereby the vigor of the tree is reduced. Complete defoliation frequently occurs in nursery stock or on young trees in the orchard. There is some difference between the appearance of the spots on sweet and on sour cherries.

Causal Organisms.[1] Karsten in 1884 described a fungus from the European bird cherry (*Prunus padus*) which he named *Cylindrosporium padi,* a name used by European workers in referring to the leaf spot disease on *Prunus* species. In America, Peck in 1878 described a fungus from wild black cherry (*P. serotina*), which he named *Septoria cerasina.* This name was generally used for some time by workers in America, although Pammel in 1889 concluded *S. cerasina* Peck should be regarded as a synonym of *C. padi* Karst. Subsequently writers referred the leaf spot organism, regardless of host, to Karsten's species. Arthur (1887), working at Geneva, New York, was the first to describe the ascigerous stage of the fungus on overwintering leaves of plum and was convinced that it was the perfect stage of *S. cerasina,* but did not name it. Finally, Higgins in 1913 and 1914 made an extensive study of the *Cylindrosporium* species occurring on *Prunus* species and described these as three separate species of the Ascomycete genus *Coccomyces.* The ones commonly occurring on *P. cerasus, P. avium,* and *P. pennsylvanicum,* he named *Coccomyces hiemalis,* those from *P. domestica, P. insititia, P. spinosa,* and *P. americana,* he called *Coccomyces prunophorae,* and those parasitic on *P. serotina, P. virginiana,* and *P. mahaleb* were referred to *Coccomyces lutescens.* Nannfeldt in 1932 changed the generic name of *Coccomyces* to *Higginsia,* for those species occurring on *Prunus* species. This reclassification has not met with general acceptance. On this basis the leaf spot generally encountered on cultivated sweet and sour cherries may be regarded as being caused by *C. hiemalis,* while that on cultivated plums is usually due to *C. prunophorae.*

Hosts. At least 18 species of *Prunus* have been reported as hosts of one or the other species of *Coccomyces* or of the imperfect *Cylindrosporium* stage. This represents most of the common wild and cultivated species of *Prunus,* including apricot but not peach.

Morphology. Stomatal infection is followed by the development of an intercellular septate *mycelium.* Oval *haustoria* are formed after the penetration of the mycelium through a constricted opening in the host-cell walls. The haustoria-invaded cells remain alive for a long period after invasion. A *stromatic* layer of cells is formed between the epidermis and the mesophyll. *Acervuli* are formed on the stroma, and from these the short conidiophores arise. The hyaline, elongated, curved or flexuous *conidia* are produced terminally on the conidiophores. These form a creamy mass which finally breaks through the epidermis and is seen as a whitish speck on the undersurface of the lesion. In some cases, due to the resistance of the epidermis to penetration, tendrils of conidia may be formed, thus giving rise to the early conception that the imperfect stage was a *Septoria.* The hyaline conidia vary greatly in size and shape depending on host relationship and other undetermined factors. Most of the conidia fall in the range of 45 to 60 by 2.5 μ, with one to two septa when mature. They are elongated, curved, or flexuous, with a tapering apex. The *apothecium* arises from the same stroma that produces the acervuli and often from the same area. The apothecium is typical of the Phacidiaceae, consisting of a black leathery hypothecium and a thinner epithecium. In this structure the *asci* are produced in early spring in the fallen leaves. These together with the paraphyses push up the covering and finally break through, forming a slit, or starlike, opening. The asci are broadly clavate 70 to 95 by 11 to 14 μ, with a stout pedicullate base. The eight hyaline continuous *ascospores* are formed in a fascicled group at the upper end of the ascus. They are linear, 30 to 50 by 3.5 to 4.5 μ. The ascospores are forcibly ejected when mature and when the leaves are thoroughly soaked.

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[1] *Coccomyces hiemalis* Hig. and *C. prunophorae,* Class Ascomycetes, Order Phasidiales, Family Phasidiaceae.

Disease Cycle. The ascocarps are produced most abundantly on leaves which fall in September and October rather than earlier in the summer. Ascospores are mature by the time the first leaves are exposed and are discharged during any rainy period for 6 or 7 weeks after petal fall. The ascospores are responsible for all primary infection, although there is some evidence that a few conidia may be produced in the old acervuli in the spring. Since the ascospores are discharged into the air at any time after the leaves have become soaked, they are readily carried by air currents or by splashing to the lower leaves of the tree. Often the first sign of infection in an orchard is on the seedlings or water sprouts close to the ground. On striking a leaf the spores become attached to the surface and, if moisture is present, germinate in a few hours. Penetration of the fungus is through the stomata and consequently entirely on the lower surface of the leaf. Young leaves, before they unfold and before developing stomata, are not susceptible to infection. The incubation period varies according to temperature. Temperatures from 60 to 68°F are most favorable for rapid development. Under favorable conditions the first evidence of infection may be as early as 5 days after inoculation, but may be delayed for 10 to 15 days at lower temperatures and in the absence of rainfall or dews. After penetration the hyphae grow rapidly through the mesophyll to the upper surface of the leaf, with haustoria forming in the host cells along the way. The purple spots, first evident on the upper surface of the leaf, are the result of the advance upward of the hyphae.

Primary infections usually are not abundant and may easily be overlooked. Very soon after the appearance of the lesions, acervuli are formed near the center on the underside of the leaf. Conidia are responsible for the extensive spread of the disease, and the rate of the build-up of leaf spotting is dependent on the periods of rainfall, since the conidia occur in sticky masses and are not easily transported except by washing or by wind-blown mist. Thus, after primary infection, if only light rains occur, there is very little evidence of the disease until fairly late in the season. There is a continuous production of conidia throughout the spring and summer, and the spores are viable after a long period of drying. Usually, new infections taper off during the hot, dry summer period. Under average conditions of rainfall the period of maximum infection and the beginning of defoliation is about halfway between blooming and harvest. Often by harvesttime in an epidemic year from 50 to 90 per cent of the leaves have dropped, but the renewed growth of the terminal shoots after harvest may give rise to another period of infection.

Control. The long period of secondary infection necessitates special attention to early control of both primary and early secondary infection. This is especially important in that defoliation prior to the ripening period reduces size and quality of the fruit in addition to exposing it to sun scald. In the nursery it is necessary to retain the foliage in order to secure a good growth of the trees during the spring and early summer.

Varietal resistance is not marked in either sour or sweet cherries. A strain of the Mazzard cherry is said to be highly resistant, and this may be useful for future breeding for resistance.

Eradicant sprays of the dinitro group have been successful in materially reducing the amount of winter inoculum when applied to the floor of the orchard about the time the ascocarps are swelling.

Summer sprays are generally effective in holding down the infection, but it has been found that some of these, especially Bordeaux mixture, has a tendency to reduce the size of the fruit, thus reducing yield and quality. Some of the low-soluble coppers have been found satisfactory for control without this reduction in size. Recent extensive trials have demonstrated the superiority of some of the organic fungicides (glyodin) as a substitute for the copper sprays. Time and frequency of application depend on weather conditions. Ordinarily, the applications should start at petal fall, with the last application immediately following harvest.

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Peach Rust

Recent investigations have indicated that there are at least three distinct but closely related rust species which attack stone fruits. In this discussion the one occurring most commonly on cultivated *Prunus* species will be treated.

History, Distribution, and Economic Importance. Very few early references have been found in horticultural writings concerning the disease, probably because of its minor importance from the standpoint of the grower. There is little question but that it was introduced into North America and other foreign countries from Europe. It was recorded as commonly occurring on peach in the United States between 1880 and 1890 and in Australia in 1890.

The rust is now world-wide in its distribution but is most commonly found in the warmer sections of the stone fruit areas. The probable reason for this will be treated under the discussion of the Disease Cycle.

Although world-wide in its distribution, the disease is of no great economic importance, partly because of the necessity of favorable climatic conditions for the development of severe infections and partly because it appears late in the season after the trees have completed sufficient growth for crop production the following year. Cases are on record of very severe damage to the crop from (1) severe fruit infections (California), (2) stunting of twig growth, and (3) defoliation resulting in devitalizing the trees. It has also been known to cause extensive and severe stunting in seedling peach nurseries, thus reducing the stand of budding stock.

Symptoms. Usually the disease does not appear until midsummer or early fall. Symptom expression varies according to the host and the severity and time of infection. Goldsworthy and Smith in their study of a severe epiphytotic of rust on clingstone peaches in California have given the most extensive description of the symptoms on leaf, fruit, and twig. The following is mainly a summary of these observations. On the *leaf* the disease is first evident as pale yellowish spots on both surfaces of the leaf. Later these spots become bright yellow. On the undersurface, rarely the upper surface, the cinnamon brown, dusty pustules of the rust fungus dot the areas. These may be scattered or so numerous as to cover the entire undersurface of the leaf, especially on plum and prune. Later, circular necrotic areas may appear on the leaf, if dry hot weather prevails. Ordinarily, however, the invaded tissue remains alive. Defoliation occurs only when the leaves are severely infected. On plum the diseased areas may take on a purple or bronze color.

The fruit is rarely infected. When present, the affected areas appear first as water-soaked dark green spots. Growth is arrested, the spots become sunken, and are deeper green than the surrounding tissue, later turning to a deep yellow. The rust pustules appear on some of these lesions, but in some cases the fungus produces internal sori which never break through the corky layer of tissue at the surface.

Twig or *bark infections* have rarely been observed on peach but are recorded as fairly common on plum in Australia and New Zealand. In the early spring when the buds are swelling, the fruiting spore pustules break through the bark without forming definite lesions. This is evidently the result of infection in the late fall of the previous year. Later, as the twig increases in size, these infected areas split open lengthwise. While most of these lesions heal over as a result of renewed bark growth, in some cases the mycelium of the fungus remains alive and will give rise to a new crop of rust spores the following year. These are mainly on the fruiting twigs, which do not make sufficient growth to heal over the lesions. Spore pustules are formed along the edge of these lesions the following spring.

Causal Organism. The rust on cultivated stone fruits is caused by a full-cycle, heteroecious rust fungus, *Tranzschelia discolor* (Pers.) Tranz. and Litv.[1] At one time the rust occurring on a number of wild species of *Prunus* was considered identical with that occurring on cultivated species, and both were classified as *T. pruni-spinosae*. Later Dunegan, as a result of extensive comparisons of collections, separated this species into two "forms," *T. pruni-spinosae typica* (mainly on wild species) and forma *discolor* mainly on cultivated species. Forma *typica* has the aecial stage on a large number of species of the Ranunculaceae, while forma *discolor* is known to occur only on *Anemone coronaria*. It is now generally accepted that the forma *discolor* should be given specific rank as proposed by Tranzschel and Litvinov in 1939 and the original specific name be retained for the rust occurring on wild species of *Prunus*. Biologic forms of the species *T. discolor* have been demonstrated by cross inoculation.

[1] Class Basidomycetes, Order Uredinales, Family Pucciniaceae.

Hosts. *T. discolor* has been reported on apricot, peach, plum, prune, almond, and cherry. In California, where only *T. discolor* is known to occur, it has been found to infect native species such as *Prunus andersoni*, *P. emarginata*, *P. fasciculata*, *P. fremontii*, *P. ilicifolia*, and *P. subcordata*, when these are grown in mixed plantings with infected apricots and peaches. The aecial stage is known to occur only on *Anemone coronaria*. Tranzschel and Litvinov suggested in 1939 that *A. hortensis* might be an alternate host.

Morphology and Disease Cycle. On *Prunus* species the *uredial pustules* are evident about midsummer but may occur earlier in warm climates. They are hypopyllous, pulverulent, and cinnamon brown in color. The *urediospores* are oblong-clavate (15 to 23 by 28 to 42 μ) and echinulate, except at the tip, which is smooth. The *teliospores* are formed in the same pustules, which now become dark chestnut brown in color. In *T. discolor* the basal cell is sparsely verrucose or smooth and is lighter in color than the apical cell. The teliospores vary from those of the genus *Puccinia* in that they consist of two distinct, easily separable cells, while the two teliospore cells of *Puccinia* are enclosed by a common membrane and are not easily separated. Teliospores measure 18 to 27 by 30 to 39 μ.

Aecia on the Ranunculaceae are much like those of the genus *Puccinia*, being cup-shaped and occurring mostly on the undersurface of the leaves but sometimes on the petioles.

The cinnamon-brown aeciospores are globoid or oblong globoid, 15 to 23 by 18 to 26 μ with verrucose walls.

Primary infection on the stone fruits may result from aeciospores or from overwintering urediospores. Thus in the warmer areas the presence of the alternate host is not essential in the life cycle. The urediospores do not survive the winter in the colder sections of the temperate zone, and since the alternate host must be in close proximity to the cultivated stone fruits, the chances of infection are greatly reduced, thus explaining the greater severity of the disease in southern latitudes. Secondary infection is through the spread of the urediospores, which are powdery and easily carried by air currents. Thus there is a rapid build-up of the disease during the late summer and early autumn months. The teliospores are formed shortly after the urediospores are mature. They overwinter, and on germination in the spring the sporidia infect the *Anemone* host. The aeciospores when mature are carried by air currents to the stone fruits, thus completing the full life cycle. There is good evidence that this rust fungus lives for some years in the perennial species of *Anemones* and will produce successive crops of spores each spring without renewed inoculation from the *Prunus* species.

Control. Stone fruit rust is seldom serious enough to warrant special control measures, especially where regular fungicidal sprays are applied for other diseases. On peach in southern California the injury at times is sufficient to warrant special spray applications.

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Other Rusts on Stone Fruits

In Asia the principal rust of stone fruits is called "white rust" because of the light color of the uredinial pustules. This rust *Leucotelium pruni-persicae* differs from *Tranzschelia discolor* in the shape and size of the urediospores and in having the echinulations uniformly distributed over the whole surface. It is widespread in Japan and China and throughout southern Asia. It has the aecial stage on various species of the Ranunculaceae. It is claimed that it occurs on the same trees on which *T. discolor* is present.

The symptoms and effect on the trees are the same as those for *T. discolor* or *T. pruni-spinosae.*

Tranzschelia pruni-spinosae (Pers.) Diet., formerly forma *typica*, occurs generally on wild species of the stone fruits, including various species of cherry. This species has the alternate host on a large number of species of Ranunculaceae, both in North America and Europe. It is absent in some regions where *T. discolor* is the common species. Much confusion has arisen in the past because this species is so similar morphologically to *T. discolor*, the main difference being in certain characters of the teliospores and the host relationships.

Coryneum Blight of Stone Fruits

Coryneum blight has other common names, "shot hole," "California peach blight," "fruit spot," "winter blight," and "pustular spot," but these are not distinctive since they have been used for other stone fruit diseases having similar symptoms.

History and Geographic Distribution. The disease was first recorded from France in 1853, but was probably present in Europe before that time since other disorders have similar symptoms. Later it was reported from North and South America, Africa, Australia, and New Zealand. It was reported as occurring in California as a destructive disease as early as 1900. In North America it is most commonly found in the western states. In the peach-growing areas of the eastern United States it is only occasionally found in neglected orchards and is of no commercial importance.

Hosts. Peach, apricot, nectarine, and sweet cherry are the stone fruit hosts on which the disease is most commonly found. It has been reported from wild cherries, European plum, cherry laurel (*Prunus laurocerasus*) and *P. davidiana*.

Symptoms. Twigs, blossoms, leaves, and fruit as well as unopened buds (leaf and blossom) are subject to attack. Variation exists among the principal hosts as to the extent to which the above organs are attacked. Wilson records that in California on peach and nectarine, dormant buds and twigs as well as leaves and blossoms are affected, but peach fruits are rarely diseased. On apricots twig infection is rare, but dormant buds, leaves, and fruit may be severely diseased. Excessive gumming of infected areas is a noticeable feature, but this may result from other causes.

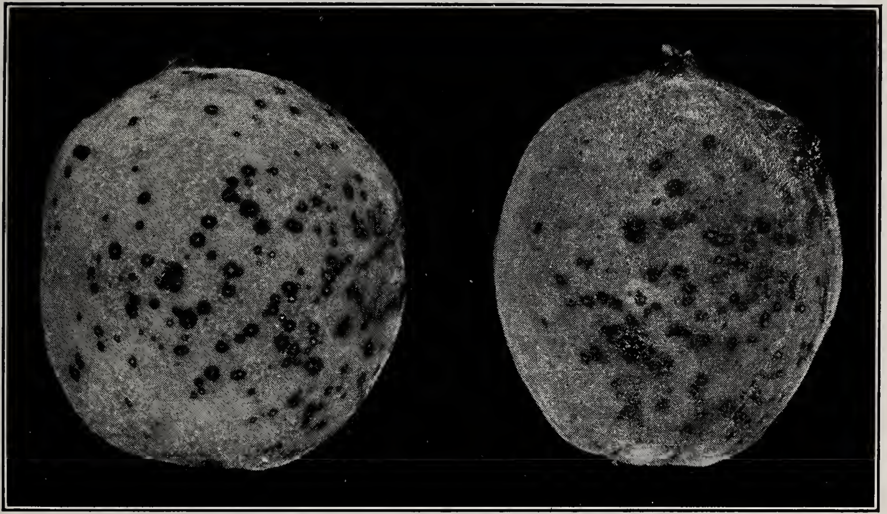

FIG. 58. *Coryneum* blight on peach fruit.

On the *fruit, Coryneum* blight lesions are seen as small, circular, deep-purple spots (Fig. 58). These spots later become raised and roughened. On *leaves* the dark brown, scattered lesions enlarge rapidly and may involve a considerable area of the leaf surface. Abscission of the diseased area follows, resulting in shot-hole effect or a ragged appearance when the lesions are large. When infection occurs on the petiole, the entire leaf is soon killed. Defoliation is not as marked as in the case of the bacterial spot.

Diseased buds are darker in color than normal ones and often show a glistening layer of gum, especially on the apricot.

Twig infection on peach results in the formation at first of small, purplish, raised spots, which later expand into elongated necrotic cankers. Severely infected twigs are killed in the late spring and early summer.

Blossom blight is the result of bud infection or of canker development at the base of the flower pedicel. The flowers wither before they are fully expanded.

Causal Organism. *Coryneum* blight is caused by *Coryneum beijerinckii* Oud.,[1] an imperfect fungus belonging in the Melanconiales. A perfect stage, *Ascospora beijerinckii*, was described by Vuillemin in France, but other investigators have failed to confirm this. The fungus produces four-to-six-celled ovoid *conidia* on short conidiophores which arise from the lesions in minute black *acervuli*. The conidia, olivaceous in color, measure 30 to 40 by 12 to 14 μ. They are not easily detached from the conidiophore by wind currents.

Disease Cycle. In the presence of moisture the conidia, when they alight on the surface of the host, become firmly attached to the tissue by the production of a gelatinous sheath. Several of the cells of the conidium may produce germ tubes which are capable of penetrating directly through the cuticle. The invading hyphae ramify in the host tissue in a limited area. From the mycelial masses finally formed in the lesions, hyphal strands push to the surface and produce another crop of conidia. According to Wilson the fungus passes its entire life cycle in or on the host.

Under California conditions where the disease is generally prevalent, the fungus is inactive during the dry hot summer months. As soon as the fall rains begin, activity is renewed and conidia are produced on the twigs and on the diseased buds. Infection of buds and twigs may occur at this time and continue throughout the winter when temperatures are above freezing. In early spring, infection of new growth occurs as the flower and leaf buds open, resulting in blossom blight and leaf infection until warm, dry weather again prevails. Thus under California conditions the critical infection period is in early winter and in the spring (or late winter) as the buds open. The conidia formed on the leaves are of little importance in the fall infection, since these have dropped before the rainy period begins.

Control. The most effective and cheapest method of control is by the use of chemical sprays. The critical time for spraying under California conditions is immediately after leaf fall, thus preventing new twig and bud infections. This spray will also control leaf curl. Bordeaux mixture is recommended for this spray because of its lasting effect during the subsequent rainy periods, when new infections might occur on unprotected twigs and buds. For peaches this one spray is sufficient, but an additional spring application is recommended for apricots. This should be applied just after the blossom buds emerge, but before petals unfold.

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[1] Class Fungi Imperfecti, Order Melanconiales, Family Melanconiaceae.

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Bacterial Canker and Gummosis

Gumming is of common occurrence on most stone fruits. Trunks, limbs, twigs, and fruit often exude gum when injured by mechanical agents or insect punctures. Cankers as a result of winter injury, insect attack, or fungus invasion also are of common occurrence. These facts have led to much confusion as to the responsible agents.

Investigations extending over the past fifty years have established the fact that some types of canker and gumming are the result of bacterial invasion. This is called "bacterial canker," or "bacterial gummosis," to distinguish it from types caused by other agents.

As in the case of fire blight, other common names have been given to this disease, depending on the organs attacked. Among these are "shoot blight," "spur blight," "sour sap," and "wither tip."

History. The first association of bacteria with gumming cankers on apricots, plums, and cherries was recorded in 1902 by Brzezinski, working at Krakow, Poland. In 1905 Aderhold and Ruhland found a bacterial canker disease causing severe damage to cherry trees in Germany and named the causative organism *Pseudomonas spongiosa*. Griffin, working in Oregon, definitely established the bacterial origin of a gummosis disease of cherry limbs in 1911. He named the organism *P. cerasus*. He stated that the bacterium produced a blight of dormant buds. Following his work Barss demonstrated that this same organism would cause cankers on limbs of other stone fruits. In 1933 Wilson published an extensive account of his investigations in California and cleared up a great deal of the confusion as to relationship of canker production, gummosis, and "sour sap" and also attempted to show that the variation in the different bacterial isolates obtained in his and other investigations could be explained on the basis of normal variations in most bacterial species. Wormald working in England between 1928 to 1940 on bacterial diseases of stone fruits, especially of plum and cherry, described various types of cankers, die back ("wither tip"), and blossom blights, which he proved were of bacterial origin. He named two species, *Ps. prunicola* and *Ps. mors-prunorum*, as responsible for these diseases. These were separated on the basis of certain cultural characters and on the fact that from trees inoculated with pure cultures of either species the same type could be recovered by reisolation. (See under Nomenclature for further details.) In 1953 Wilson summarized the research on this disease (see references).

Geographic Distribution. Stone fruit canker and gummosis have been reported from northern and western United States, the British Isles, Holland, France, Germany, Denmark, and Australia. In North America the stone fruit canker disease is of most importance in the Pacific Coast states but has been reported from various eastern states. If *Ps. syringae* is considered as the organism concerned, the distribution of this species on its various hosts probably is world-wide.

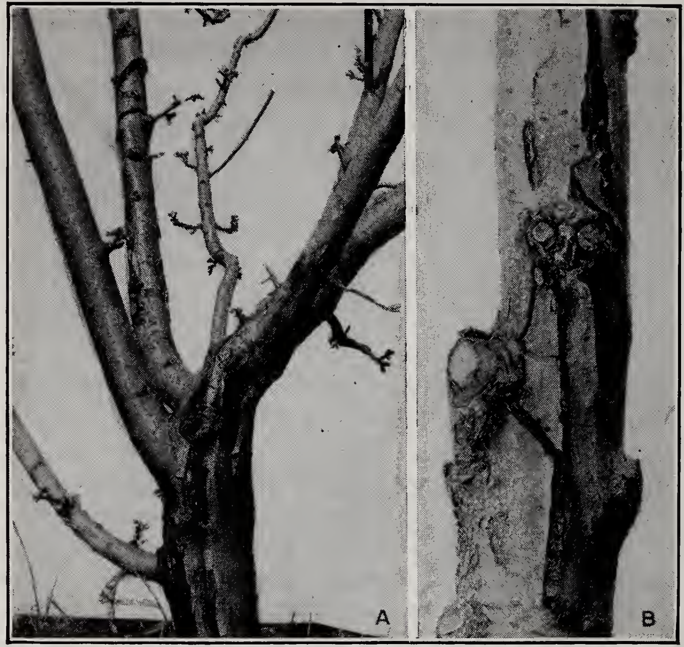

FIG. 59. Bacterial canker, or gummosis. *A*, canker on plum; *B*, cracked and raised bark on plum canker. (*Calif. Agr. Expt. Sta.*)

Symptoms. Symptom expression is quite variable. This variability is due to variation in climatic conditions between regions where the disease occurs and to other reactions of different species to the invasion of the bacteria. Trunks, limbs, shoots, fruit spurs, blossom, dormant buds, leaves, and fruits are subject to attack. No invasion of the root system has been reported. In most host species the most destructive phase is that on the *trunks* and *branches* (Fig. 59). Circular to elongated, water-soaked lesions appear in the bark and outer sapwood. The invaded bark is brown, gummy, and usually sour-smelling. When branches are girdled they either fail to develop in the spring or die in the summer, following some early leaf development.

Blighting of dormant buds is common on cherry and apricot. Small cankers are produced at the base of the diseased buds.

Blossom blight is reported as occurring in California, most commonly on plums and cherries. In England blossom blight has been reported as common on pears.

Leaf spotting is said to be common in England on plum and cherry leaves, but is rather rare in California owing to the prevailing dry conditions during late spring and summer when leaf infection occurs. The leaf spots consist of small purple lesions similar to those found on the peach and plum caused by *Xanthomonas pruni*. These are more commonly seen on plum and apricot but are rarely observed on peach. A shot-hole effect is produced by the early excision and dropping out of the diseased tissue. Defoliation is not as extreme as in the case of *X. pruni* attacks.

Fruit invasion results in sunken black lesions on apricot and cherry.

Shoot withering is recorded as a common symptom in England, while in California it is reported as occurring mainly on a few varieties of plum. Under English conditions the bacteria invade the older twigs, while in California the downward extension of the bacteria is soon halted and little damage results.

Causal Organism. *Nomenclature.* In 1905 Aderhold and Ruhland named a bacterium causing a serious canker and gummosis condition of cherry trees in Germany *Bacillus spongiosus*. Griffin in 1911 named the cherry gummosis organism *Pseudomonas cerasus* (*cerasi*). Wormald called the green shoot wilt organism *Ps. prunicola*, and later another bacterium differing in some cultural characters he described as *Ps. mors-prunorum*. Wilson isolated two distinct types of bacteria from cankers in California. One produced a green or fluorescent color in the medium, while the other was described as "white," but producing a yellow color on aging. Because this organism was similar to *Ps. cerasi* in its pathogenic characters he named it *Ps. cerasi* var. *prunicola* n. var.

The species here listed while producing much the same symptoms may be separated into two groups: (1) those producing a green or fluorescent pigment and (2) those having generally a white type of growth with a tendency to the production of a yellowish pigment on certain media on aging. In Elliott's revised edition of her "Manual of Bacterial Plant Pathogens" (1951), she lists all of these species with the exception of *Ps. mors-prunorum* under *Ps. syringae* Van Hall. This is a widely distributed organism which under a number of different names has been reported on some 80 hosts, including 11 species of *Prunus*.

Included in the diseases caused by this composite species are lilac blight, citrus blast, pear blossom blast (not fire blight), and a number of legume diseases. This combination is justified on the basis of similarity of cultural and morphological characters and on the cross-inoculation studies carried out by a number of workers in recent years. Some gaps exist in the cross-inoculation studies, and some differences in pathogenicity are noted, but these differences are not sufficient to warrant species separation. Thus *Ps. syringae* Van Hall[1] is now the accepted name for bacterial canker and gummosis disease of stone fruits.

Ps. mors-prunoram Wormald is regarded as a valid species differing from *Ps. syringae* in the lack of production of the green fluorescent pigment and in being positive to the Gram stain.

Hosts. Included in the 80 hosts of this bacterium are the following stone fruits: *Prunus persica, P. avium, P. cerasus, P. domestica, P. mahaleb, P. armeniaca, P. pumila, P. salicina, P. salicina × munsoniana,* and *P. simonii.* Of these the peach is the least susceptible, while sweet cherries are the most susceptible, followed by some varieties of plum.

Morphology and Disease Cycle. The rod-shaped bacterium measures 0.6 by 1.2 to 1.8 μ. It does not form spores, is capsulate, motile by one to several polar flagella, and is Gram-negative. It produces a green fluorescent pigment in culture.

Infection apparently takes place during the fall months. The infected areas appear as small brown flecks in the cortex of the bark. Under California conditions some of these areas enlarge and become evident as gumming cankers as early as January and continue to appear throughout the spring months. During the summer the bacteria are quiescent, but resume activity in the fall, when they extend into the adjoining live tissue in brownish streaks. At the same time the bacteria are washed down to give rise to new infections. In early spring the tissue between the brown streaks becomes necrotic and the tissues turn "sour." If infection takes place too early in the fall the bacterial masses are effectively walled off and fail to develop cankers in the spring.

In England it has been proved that infection usually occurs through the leaf scars shortly after abscission. Infection of leaves and blossoms occurs as soon as these expand in the spring.

Relation to Environment. Bacterial canker and gummosis is most prevalent in England and in the Pacific Coast region of the western United States. It has rarely been reported from the northern Middle West states. It would appear, therefore, that mild winters and moderate summer temperatures are most favorable to the disease.

Species and Varietal Susceptibility. On species of cultivated stone fruits the apricot is regarded as most susceptible. Plums (*Prunus domestica* and *P. salicina*) and sweet cherry (*P. avium*) rank second. Peach and nectarine are least susceptible, while almond is rarely attacked. On plums used as rootstocks the Myrobalan is much less susceptible than the Marianna hybrid. Resistant strains of the Myrobalan plum and Mazzard cherry have been reported from England. Among commonly grown plum varieties in California, Duarte, President, Wickson, and Burbank are reported as "tolerant" to the disease (in increasing order). All varieties of apricot commonly grown are regarded as very susceptible.

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[1] Class Schizomycetes, Order Eubacteriales, Family Pseudomonadaceae.

Control. Where practical, varieties of plum and cherry resistant to the disease should be selected. It has been found that plums grafted or budded on peach rootstock are cankered much less than those on their own roots or on plum rootstock.

Since infection occurs in the fall, bactericidal sprays such as Bordeaux mixture should be applied in the fall at about the time the first leaves are dropping. Spring spraying is also necessary to prevent leaf and blossom infection where this type of infection is common.

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Silver Leaf of Stone Fruits

Although this disease is of considerable importance on apples (page 180), it is more generally found on stone fruits and consequently will be treated as a disease of this group.

History. The first report of the disease was by Prillieux in France in 1885. He noted the leaden luster of the leaves and showed that it was due to air spaces between the upper epiderminal cells and the palisade parenchyma. It was also noted by Sorauer in Germany (1886) and by Aderhold (1895). These authors considered it to be of nonparasitic origin.

In 1902 Percival in England gave it the name of "silver leaf" and established the parasitic origin of the condition by demonstrating the presence of the fungus hyphae in the wood, and by inoculation brought about the silver leaf symptoms. Subsequent investigations by English and Canadian pathologists not only established that the fungus *Stereum purpureum* was the cause, but worked out many details in the life cycle of the disease and its true relationship to the silvering effect. It was pointed out by these and other workers that a silver leaf condition may be due to agents other than this fungus, but that the "true" silver leaf is always associated with the presence of the fungus in the wood of the tree even where the fruiting stage of the fungus is not evident on the exterior of the trees.

Geographic Distribution. Silver leaf has been reported from most countries of continental Europe and is especially common in England. In New Zealand it is general in all the fruit sections and has been reported from South Africa. In Canada a survey demonstrated its presence on apples from Nova Scotia to British Columbia. Reports from the United States show that the disease is of most importance in the Northwest, but that the fungus has been collected on various hosts throughout the country. It is not regarded as of any importance in most eastern sections of the United States. It should be stated that not all the reports of silver leaf have been confirmed by demonstrating the presence of *Stereum.* Winter injury and other agents will give similar symptoms.

Hosts. Of the stone fruits certain varieties of plums are most severely affected, but it has been recorded on sour cherries, apricots, almonds, and peaches. In addition it is commonly reported on apples, currants, and gooseberries, as well as many other hosts both cultivated and wild. The fruiting bodies of the fungus may be found on many forest trees, but probably at best it is only a weak parasite on these hosts.

Symptoms. The first evidence of the disease is on the foliage, which shows a metallic luster, in marked contrast to the normal green of healthy trees. This symptom may not appear, however, until the tree has been diseased for some time.

A cross section of the limb of a diseased tree will show a dark discoloration of the heartwood where the fungus is growing. The darkened areas may involve most of the heartwood or consist of only small streaks. When sections of this diseased wood are set upright in water in a moist chamber for 48 hours, there is a marked diffusion of the brown color into the water, and a white cottony growth of the fungus develops on the surface exposed to the air.

Death of the infected branches or of the entire tree results within a year or so after the appearance of the silvered leaves. Stunting of growth is often apparent before the tree dies.

The fruiting bodies characteristic of the disease may appear on the surface or from cracks in the bark before the death of the tree, but may be entirely absent throughout the progress of the disease. These at first consist of a flattened plate, circular or irregular in form, with a faint purple color. They may vary greatly in size from ¼ to 1 or more inches in diameter. Later these take on a shelflike (imbricated) form. They vary in color from a very faint to deep-dull purple. The fruiting bodies may be found on limbs and trunks of trees after the tree is entirely dead.

Causal Organism and Disease Cycle. Silver leaf is caused by *Stereum purpureum* Pers.[1] The thin leathery *sporophores* of the silver leaf fungus are variable in size and form. They may appear as small circular resupinate bodies about ¼ in. in diameter. They may remain flat on the surface or form an imbricated shelving structure up to 1 in. in diameter. The purple *hymenium* (fruiting layer) is smooth, and the basidia extend over the entire surface. The *basidiospores,* borne on four sterigmata on the basidia are hyaline, 5 to 7 by 2.5 μ. Cystidia are mingled with and extend above the basidia. The upper surface of the shelving sporophores is villose tomentose, buff or grayish in color. The basidiospores are forcibly discharged and may travel for some distance in air currents.

The fungus is a wound parasite, usually entering through pruning wounds. Following infection the fungus penetrates the heartwood. It grows through the vessels, advancing upward more rapidly than downward, and may finally penetrate the sapwood. A gummy substance is formed in the vessels, thus restricting the movement of water and solutes. The exact cause of the silvering effect on the leaves is not known, but is definitely not due to the invasion of these organs by the fungus.

The final killing effect is caused by the invasion of the sapwood. After this invasion the fungus may grow out through the bark and produce the characteristic sporophores. Whether or not sporophores are produced depends on favorable factors such as abundant moisture and reduced vigor of the tree.

Relation to Environment. Silver leaf appears to be most prevalent in regions having mild open winters, although in Canada where the disease is prevalent on apples, winter injury is suggested as a predisposing factor. In regions in England where peaches are grown under glass, silver leaf is a serious disease, thus contradicting this supposition. It is not a common disease in eastern United States on either apples or stone fruits, although the fungus is generally distributed over this area. This would indicate that vigorously growing trees under favorable climatic conditions are able to withstand any infection which might occur.

Control. Good cultural practices seem to be indicated for the control of silver leaf. Large pruning wounds should be avoided, and all brush which might harbor the fungus should be removed from the floor of the orchard. The trees should be kept under the best possible growing conditions.

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[1] Class Basidiomycetes, Order Agaricales, Family Thelephoraceae.

In areas where the disease is known to be prevalent, the wounds should be treated with some disinfectant such as Bordeaux paste.

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Verticillium Wilt of Stone Fruits

Verticillium wilt is of considerable economic importance on stone fruits throughout the Temperate Zone. Since the symptoms and effects on tree fruits differ somewhat from those on herbaceous crops and cane fruits, a brief account of the disease as it affects stone fruits is presented. For a full discussion of this disease, see *Verticillium* Wilt of brambles, page 318.

Geographic Distribution. *Verticillium* wilt on stone fruits has been reported from most regions of the temperate zone. According to Rudolph, the disease is seldom found in the United States below latitude 35.

Symptoms. "Wilt" is not a proper descriptive term for the disease on woody plants, since there is seldom any evidence of flaccidity. The first evidence of the disease is the defoliation on the affected limbs in early summer. The leaves show a slight blanching and have a dull appearance before dropping. In tree fruits as well as brambles, the disease progresses upward, so that the lower leaves of the branches are first affected. The whole tree may be affected, or the disease may be confined sharply to one side or one limb of the tree.

The trunk, branches, and twigs show no external evidence of the disease other than a general stunting. Dense sclerotia of the fungus may appear on dead twigs during the winter. In cross sections of the stem the diseased tissue is evident as a ring of discolored wood, gray or light or dark brown in color. Sometimes the ring is discontinuous or confined to a definite segment. In longisection the diseased wood is evident as streaks of discolored xylem. Roots show much the same symptoms as the stem, but since the fungus invades the roots first, the discoloration is usually more intense, often showing as grayish black in some segments.

Diseased trees are not always killed, but may remain stunted and unproductive for several years.

Causal Organism. The cause of *Verticillium* wilt is *Verticillium albo-atrum.* This fungus is fully described under *Verticillium* Wilt of brambles, page 318.

Disease Cycle. *Verticillium albo-atrum* is a soil-inhabiting fungus, and invasion takes place through the root system. The fungus progresses upward through the xylem vessels. Lateral spread to neighboring tissues occurs after the general invasion of the xylem. The disease cycle is much the same as that described for this disease on brambles (page 318).

Hosts. All species of stone fruits have been reported as subject to this wilt. As indicated before, the fungus attacks several hundred host species in many genera. In California, Rudolph reports that Myrobalan seedlings are especially susceptible, thus posing a problem in nurseries where this species is used for budding stock.

Selected Reference

Rudolph, B. A.: *Verticillium* hadromycosis. *Hilgardia,* **5**:197–353 (1931).

See also references under Brambles.

Clitocybe Root Rot of Fruit Trees

History and Distribution. A root rot very similar to that caused by *Armillaria mellea* is caused by a closely related fungus *Clitocybe tabescens* (Fr) Bres.[1] The fact that this fungus rather than *A. mellea* was responsible for the root rot of apple, cherry, and peach trees in Oklahoma was pointed out as early as 1901 by Wilcox. Recently it has been extensively studied by Rhoads and others, especially in the peach orchards of southeastern United States.

While *A. mellea* is widely distributed throughout the Temperate Zone, *C. tabescens* and the root rot caused by it seem to be much more common in southern and subtropical areas, although the fungus has been collected in more northern latitudes.

Losses from this disease have been extensive, at least in the southeastern section of the United States. This loss has increased in those sections where old orchards are replanted to peach. Growers complain that the present orchards have a shorter life than was formerly the case. No exact estimate of loss is possible, since few surveys have been made by those able to distinguish this disease from other conditions such as winter injury and *Armillaria* root rot. A few accurate surveys indicate that from 40 to 60 per cent of replants in old orchards where this disease has become established are killed within 10 years.

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[1] Class Basidiomycetes, Order Hymeniales, Family Agaricaceae.

The gross *symptoms* of this disease are identical with those described on page 255 for *Armillaria* root rot. Some differences in the character of the rhizomorphs are suggested by Rhoads. The black "shoestrings" are likely to be less conspicuous and the white, fan-shaped plates between the bark and the wood are less in evidence. He also calls attention to a peculiar perforated appearance on the platelike mycelial growth when the bark is stripped from the diseased area.

The *disease cycle* is much the same as that of *A. mellea*. Like this species, the infection, under favorable conditions, can take place through the uninjured bark, and the relation of roots to the survival of the fungus in the soil is much the same.

The *fruiting bodies*, the "toadstools," are found developing at the ground line, usually around trees which are dead or in the last stages of the disease. These are often absent, especially under dry soil conditions or where the peach trees have been mounded. The *sporophores* arise from a basal growth at the ground line and are usually in clusters of a few to many individuals. The *caps* are convex to flattened, whitish to light tan, smooth or with tufts of fibrils near the center, and from 2 to 3½ in. in diameter. The *gills* are white. It differs from *A. mellea* in the absence of an annulus on the stem and in being more slender in growth. The fruiting bodies are produced in the fall and soon decay, or if dry weather prevails they may dry up and turn black but remain recognizable for several weeks.

The spread of the disease probably is due to the growth of the fungus mycelium through the soil, although there is reason to believe that new infections arise from the spores which are produced in great abundance on the gills of the fungus.

Control methods are much the same as for the *Armillaria* root rot. Resistant varieties of peach have not been found to date. Since this is primarily a root disease, it would be necessary to find a suitable resistant stock on which the standard varieties of peach could be budded.

The host range of this fungus is very extensive. It is known to attack 210 species of plants in 137 genera. Thus it would be difficult to select a site on recently cleared land with any assurance that the disease would not appear. Orchards where trees have died as a result of the attack of this fungus should not be replanted to peach unless every effort has been made to remove all roots of the old trees. (See *Armillaria* Root Rot for further control measures.)

Selected Reference

Rhoads, A. S.: *Clitocybe* root rot found wide spread and destructive in Georgia and South Carolina peach orchards. *Plant Disease Reptr.*, **38**:42–46 (1945).

———: A comparative study of two closely related root rot fungi, *Clitocybe tabescens* and *Armillaria mellea. Mycologia,* **37**:741–766 (1945).

———: *Clitocybe* root rot of woody plants in the southeastern United States. *U.S. Dept. Agr. Circ.*, **853** (1950).

Wilcox, E. M.: A Rhizomorphic root rot of fruit trees. *Okla. Agr. Expt. Sta. Bull.*, **49** (1901).

Armillaria Root Rot of Stone Fruits

Armillaria root rot, caused by *A. mellea,* is treated in detail under diseases of pome fruits (page 133). As stated in that treatment, it is of importance on all fruit crops.

This disease is widely distributed throughout the stone fruit–growing areas of North America. It is more commonly found on peach trees than on other stone fruits and is often prevalent in orchards following the clearing of the land of forest trees or old apple orchards.

The *symptoms* on the peach may be confused with winter injury or water logging. The first year of evident trouble, the tree takes on a weak appearance with small yellowish leaves over the entire tree or confined to one or two branches. At this time there is little aboveground evidence of dying of the trunk or limb tissues. The entire tree or single branches may die by the end of the summer. If the tree survives the winter, it is likely to die the following year. Often diseased trees are grouped in one area in an orchard.

When the roots are exposed, it is possible to differentiate this root rot from winter injury by the location of the dead areas on the roots. In *Armillaria* root rot, the disease lesions are generally near the union of the larger lateral roots with the main crown root. Winter injury is more likely to be on the trunk at or below the ground line. In addition to the location, the presence of fan-shaped plates or strands of the fungus is a distinguishing feature of the disease. However, these symptoms are much the same as for *Clitocybe* Root Rot.

Control. See *Armillaria* Root Rot of pome fruits. In California the carbon disulfide soil treatment is generally favored. This may be due in part to the fact that certain injurious insects and nematodes are killed by this treatment.

Minor Disease of Stone Fruits

Fusicoccum Canker of Peach. This leaf spot and canker of peach has been reported from Europe and the United States. It appears to have become of increasing importance recently in the North Atlantic Coast area. The pathogen *Fusicoccum amygdali* Delacr. was originally described as occurring on almond by Delacroix (1905) in France. In 1941 Haenseler and Daines reported it for the first time in the United States, but they state that it had been observed in New Jersey several years previously. In 1940 it was found in Massachusetts, and Guba reported in 1953 that it was general in the North Atlantic Coastal area.

The disease affects the leaves and twigs and is destructive on certain peach varieties, notably June Elberta, Belle of Georgia, Golden Jubilee, and Greensboro.

The spots on the *leaves* are large, irregular or circular in outline, often zonate and brown in color, with scattered black pycnidia near the center.

The cankers appear in the current season around the buds on wood of the previous season's growth. Auxiliary infection occurs throughout the growing season. Infections late in the season often do not show as cankers until the following spring. The buds on infected twigs are killed before any growth takes place. Girdling of the twigs at their base results in the death of the shoots after growth starts in the spring.

The *cankers* formed at the nodes resemble those produced by the brown rot fungus or by spray injury. Gumming is common but is not characteristic of *Fusicoccum* canker, since several other canker diseases of the peach exhibit gum exudation. The black *pycnidia* appear over the surface of the cankered areas. The *conidia* ooze from these pycnidia in long tendrils under moist conditions and furnish inoculum for continuous infections throughout the season. The fungus overwinters in the twig cankers.

The pycnidia are large and often chambered. The conidia are single-celled hyaline and fusoid. No perfect stage has been found. In regions where this disease is prevalent control is accomplished by frequent fungicidal sprays throughout the growing season. In vigorous well-cared-for orchards the disease is usually of minor importance.

Selected References

Delacroix, G.: *Fusicoccum amygdali* Delacr. *Bull. Soc. Mycol. France,* **21**:180–185 (1905).

Guba, E. F.: Large leaf spot and canker of peach caused by the fungus *Fusicoccum amygdali* Delacr. *Plant Disease Reptr.,* **37**:560–564 (1953).

Haenseler, C. M., and R. H. Daines: Peach canker caused by *Fusicoccum amygdali* Delacr. (Abstr.), *Phytopathology,* **31**:10 (1941).

Powdery Mildew of Peach and Apricot. Although not a disease of major importance, powdery mildew of the peach and apricot may be of considerable importance where weather conditions are favorable for infection. For example, Fikry reports it as one of the most serious diseases of peach in Egypt, causing decided reduction of fruit and marring of that produced, so that it has to be sold at a much lower price. It is often a serious problem in nurseries where the seedling stocks are often stunted when attacked early in the growing season. On apricots in California, Yarwood reports a serious loss as a result of rejection of marred fruit by the canners.

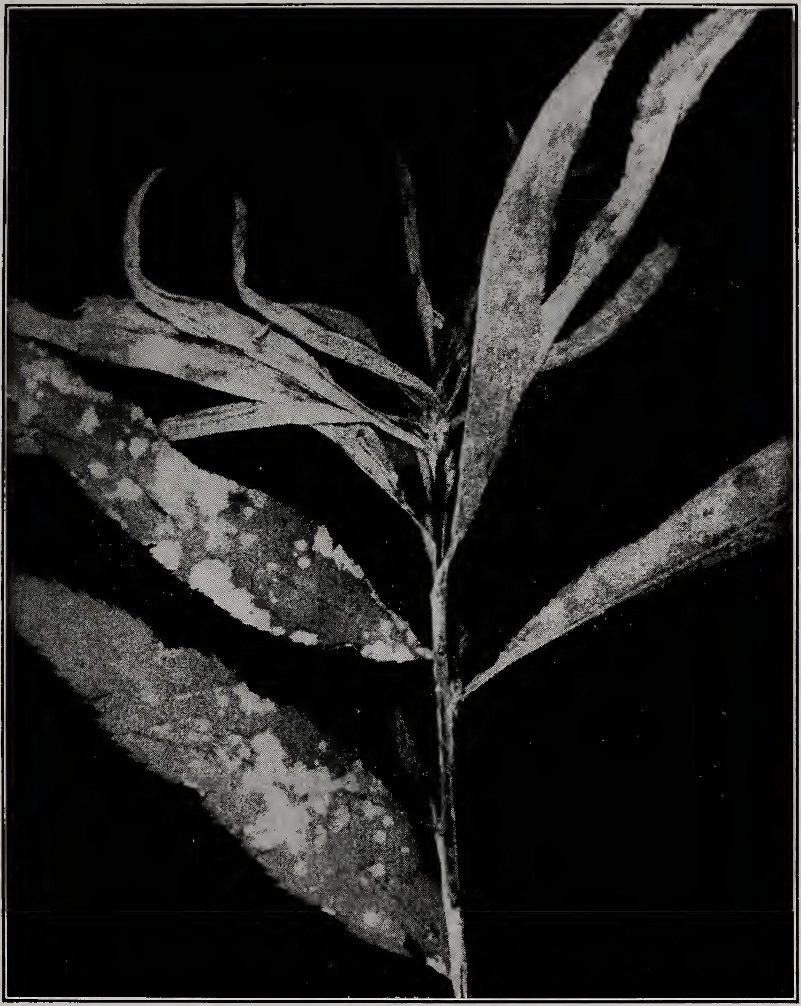

Fig. 60. Powdery mildew peach. Upper leaves dwarfed and curved, lower leaves with spotted white mildew.

Two species of the powdery mildew fungi attack the peach. The most common and destructive one is *Sphaerotheca pannosa* (Wallr.) Lev., ordinarily known as the "rose mildew." *Podosphaera oxyacanthae* the common species on cherry also is recorded as occurring on peach, but is much less common and does little damage.

S. pannosa attacks leaves, young shoots, and fruit. The young leaves may become entirely coated with a thick layer of the mycelium and are curled and narrowed as they expand (Fig. 60). On older leaves large white patches with radiating edges are the rule. The white, powdery layer may extend over the entire terminal portion of the growing shoot, or individual patches as on the older leaves may appear. On the *fruit* the disease first appears in the form of white round spots which increase in size until a large portion or the whole surface of the fruit is involved. The fruit takes on a pinkish color, which later turns to a dark brown. The epicarp of the fruit becomes leathery and hard, sometimes cracking. If it does not fall off, it is rendered worthless. This is the condition where powdery mildew thrives, as in Egypt, but it is not so common on fruit in most regions even when it is present on the leaves and shoots. On the apricot fruit the fungus produces small necrotic areas which give the mature fruit a "dirty" appearance.

The white, powdery appearance is caused by the great numbers of *conidia* produced in chains arising from the wefts of hyphae on the leaf surface. These are produced rather early in the season and give rise to secondary infections. Older leaves and fruit are fairly resistant to infection. The perfect stage is rarely found on peach and apricot but is common on such hosts as the rose. The *perithecia* have hairlike, unbranched flexuose appendages and they contain a single *ascus* with eight *ascospores*.

There is some question as to whether the fungus on peach is properly referred to as *S. pannosa*. Some authors regard it as a special strain of this species. However, Yarwood in California submitted evidence that infections on apricots were from mildewed roses in the neighborhood of the apricot trees and that when these were removed the mildew on the apricots did not appear. He was able to infect apricot fruit with conidia from rose. He pointed out, however that there were probably four strains of this mildew giving different reactions on apricot. One strain attacks both peach and apricot, while one from rose does not attack apricot at all. Since it is known that peach mildew can appear in the absence of the disease on roses, there must be other explanations for initial infection. It may be that the fungus is able to live over in the peach buds, as is the case with the powdery mildew of apple, but there is no evidence on this point.

Powdery mildew of peach can be controlled by applications of sulfur fungicides, but where regular sprays are applied for other peach diseases it is rarely necessary to make any additional applications for this disease.

Selected References

Fikry, Amin: The control of peach mildew. *Ministry of Agr. Egypt Tech. Sci. Service Bull.,* **183,** (1937).

Yarwood, C. E.: Apricot mildew from rose and peach. *Calif. State Dept. Agr. Bull.,* **41**:19–25 (1952).

Powdery Mildew of Cherry. Aside from infections on nursery stock, powdery mildew is not an important disease, although at times serious outbreaks occur in the nursery planting and seriously stunt the terminal growth. In general, budded sour cherry stocks are most severely attacked, but Mazzard stocks and sweet cherry varieties may suffer some damage.

The disease is caused by *Podosphaera oxyacanthae* (D. C.) DeBy, one of the common species of the powdery mildew group of fungi. This same species has been reported from such cultivated plants as plum, peach, apricot, apple, pear, quince, hawthorn, serviceberry, spirea, and persimmon.

The fungus attacks young twigs and leaves. The general effect is upward curling of the leaves, with the terminal leaves decreasing in size and a shortening of twig growth. The mycelium grows rather evenly over the surface of the leaf and shoot but sometimes appears in patches. With the production of the conidia on this mycelium the characteristic powdery coating results. Secondary infections from these conidia occur throughout the season when weather conditions are favorable. Later in the season numerous black *perithecia* appear, especially on the underside of the leaf. The perithecia have appendages dichotomously branched and recurved at the tip. Each perithecium contains a single *ascus* with eight *ascospores.* These perithecia carry the fungus over winter on the fallen leaves, and the ascospores are released to bring about initial infection in the spring. As is the case with most powdery mildews, hot dry weather with sufficient moisture in the form of high humidity, fogs, dews, or intermittent rains, to permit the germination of the spores, seems to be the best environment for the development of the disease.

Where powdery mildew is a serious disease, as in some nurseries, control can be obtained by rather frequent applications of sulfur dust or liquid sprays of wettable sulfur. Where sprays are applied for the control of cherry leaf spot, this disease is not likely to appear.

Selected Reference

Stewart, V. B.: Some important leaf disease of nursery stock: powdery mildew of cherry. *Cornell Univ. Agr. Expt. Sta. Bull.,* **358**:192–194 (1915).

Powdery Mildew of Plum. The same fungus, *Podosphaera oxyacanthae,* causing cherry mildew, occasionally attacks the plum but is rarely severe enough to cause any damage, and control measures are rarely required. For details see Cherry Powdery Mildew.

Perennial Canker, Die Back, and Gummosis of Stone Fruits. Cankers, especially on peach, have been ascribed to various agents, including several fungi such as *Sclerotinia fructicola, Fusicoccum amygdali, Valsa leucostoma,* and *Valsa cincta.* Die back of weakened twigs of the peach is usually ascribed to *Valsa leucostoma* since the fruiting bodies of this fungus are commonly found in the dead twigs, although the fungus may not have been the original cause of dying. The common production of gum exudate on the surface of the cankers of stone fruit trees has given rise to the common name "gummosis," but many agents may be responsible for the initiation of gum flow, and consequently this term is too general.

Perennial canker of peach usually is the result of wound infections by two species of *Valsa, V. cincta* Fr. and *V. leucostoma* (Pers.) Fr. These two fungi are considered weak parasites and are rarely of importance in well-cared-for orchards, except under conditions where winter injury occurs. After these fungi are established in the dead tissue, they may persist, and by repeated invasion of the live tissues around the wound cause extensive cankers. Weak twigs or those injured by low winter temperatures frequently are invaded by one or the other of these species, as evidenced by the numerous pycnidia observed on the twigs in the spring.

The first external evidence of the canker is the appearance of gum drops around a wounded area in the bark. The bark tissues of the invaded area at first are somewhat slimy but later dry out and collapse. During the summer the numerous pycnidia appear over the surface as pimples. The pycnospores are exuded in long amber-colored tendrils during wet periods. When growth is resumed in the spring a callus layer is formed around the invaded area, but as growth declines during the autumn, the fungus renews invasion and advances beyond the callus barrier. In some cases the invading fungus stops growth and the production of callus more or less closes the wound.

The imperfect stage of these fungi is a *Cytospora,* and it is this stage which commonly appears on the surface of the cankered area. The perfect or *Valsa* stage is less commonly found, but in some years it may be abundant.

Of the two species of *Valsa, V. cincta* is regarded as most pathogenic, especially in northern latitudes. It is rarely of importance in the southern peach-growing regions.

Control. Since *Valsa* is a wound parasite, it is advisable to follow practices in pruning that will avoid producing large wounds. The fungus rarely invades smaller wounds when these have a chance to heal rapidly. Cankers should be cut out as soon as observed.

Selected References

Hildebrand, E. M.: Perennial peach canker and the peach canker complex in New York, with methods of control. *Cornell Univ. Agr. Expt. Sta. Mem.*, **276** (1947).

Willison, R. S.: Peach canker investigation, I. *Sci. Agr.*, **14**:32–47 (1933). II. *Canadian J. Research,* **14**:27–44 (1936). III. *Ibid.*, **15**:324–339 (1937).

Rhizopus Rot ("whiskers"). A serious rot of stone fruits, especially peaches in transit and storage, is caused by the bread mold fungus *Rhizopus nigricans.* In 1925 Anderson found that it was more frequently reported by fruit inspectors as the cause of rot in transit and market than was brown rot. The fungus spreads rapidly through the container. Often a single infected peach results in the rotting of most of the fruit within a few days if the temperature is above 50 or 60°F. The common name "whiskers" is derived from the fact that the fruit is covered with the mycelium of the fungus, and the black fruiting bodies extend out into the spaces between the fruits and even on to the sides of the container. In recent years the damage from this rot has been greatly reduced by care in handling the fruit. The fungus is a wound parasite, and consequently, care should be taken to avoid any break in the fruit surface. Also, as the fungus requires rather high temperatures for rapid growth, precooling and maintaining low temperatures in transit and market will prevent its rapid spread through the container.

Overripe fruit should not be included in the container since such fruit is subject to bruising and subsequent infection.

The fungus *Rhizopus nigricans* is fully described under *Rhizopus* Rot of strawberries. [H. W. Anderson, *Rhizopus* rot of peaches. *Phytopathology,* **15**:122–124 (1925).]

CHAPTER 7

DISEASES OF DRUPE OR STONE FRUITS (CONTINUED)

VIRUS, NONPARASITIC, AND NEMATODE DISEASES OF STONE FRUITS

Stone fruits, in contrast to pome fruits, are affected with an unusual number of virus diseases. Prior to 1930, only five virus diseases of stone fruits had been recorded, and all these affected peach. Since that date, at least 45 more have been added, of which 20 occur on the cherry group, 5 on plums and prunes, in addition to a number of new ones reported on the peach group. Furthermore, it has been found that many of these diseases have a much wider host range than was formerly thought. Thus when a particular disease, for example, is listed as "peach rosette," it is not implied that the disease occurs exclusively on this host, but merely that it is of greatest significance or was first described on this host.

The staggering number of "new" virus diseases described does not mean that these have suddenly appeared from "the blue," but rather that more intensive research has resulted in a better understanding of this group of diseases. Also, it is probable that some have long been present on wild hosts or on symptomless carriers and have appeared in the orchards when favorable conditions arose for their dissemination. It is likely that further investigation of the many virus diseases of stone fruits will establish the fact that two or more may be due to the same virus and thus reduce the number previously described.

Stone fruit virus diseases, on the basis of symptom expression, represent several types. Most of the important peach maladies fall in the "yellows" class, but phony peach is distinctly a "dwarfing" type. The "mosaics," "ring spots," and "mottle" types are more commonly found in the cherry and plum groups.

Most of the stone fruit viruses can be transmitted only by some form of tissue grafting. Natural spread by insect vectors is known in a few and suspected in most. One at least is known to be seed-borne, while another is suspected of being soil-borne.

Recently, some success in transmitting some of the stone fruit viruses to young cucurbit plants has developed a technique which may be useful in differentiating the entities involved in some of the diseases which seem to result from the presence of more than one virus acting on the host.

Nomenclature of Viruses. A number of systems for naming viruses and virus diseases have been suggested, but at present no universally acceptable system has been adopted. In the treatment of the stone fruit virus diseases which follows, it was found difficult in some cases to select acceptable names for either the disease or the virus. This was especially true where it was thought best to group several diseases under a "complex." The nomenclature and synonymy of some of the viruses and diseases treated in this text are presented in Table 2, below, for the convenience of the reader. The names are, in part, those used in *Agricultural Handbook* 10, but the author is responsible for the combinations in the "complex" groups.

In the following pages only a few of the many stone fruit virus diseases are treated in detail. Those of less importance are treated in such a manner as to bring out the salient facts. For further detailed information the reader is referred to "Virus Diseases and Other Disorders with Virus-like Symptoms of Stone Fruits in North America," published as *Agricultural Handbook* 10 by the U.S. Department of Agriculture in 1951.

TABLE 2. SYNONOMY OF STONE FRUIT VIRUS NOMENCLATURE*

Common names of diseases	*Names of viruses*
Peach yellows	*Chlorogenus persicae* Holmes Peach virus 1 (Johnson) *Prunus* virus 1 (Kunkel) Smith
Peach rosette	*Nanus rosettae* Holmes *Carpophthora rosettae* Holmes Peach rosette virus Peach virus 2 Johnson *Prunus* virus 2 (McClintock) Smith
Little peach (Peach littles)	*Chlorogenus persicae* var. *Micropersica* Holmes Peach virus 3 Johnson *Prunus* virus 1A Smith
X-disease complex (X-disease) (Eastern X-disease) (Yellow-red virosis) (Western X-disease) [Buckskin (Cherry)] (Little cherry) (Peach leaf casting yellows)	*Marmor lacerans* Holmes *Carpophthora lacerans* (Holmes) McKinney Buckskin virus (Cherry) Western X-disease virus Little cherry virus

* Based on *U.S. Dept. Agr. Handbook,* **10** (1951).

Table 2. Synonomy of Stone Fruit Virus Nomenclature (*Continued*)

Common names of diseases	*Names of viruses*
Phony (Phony disease) (Phony peach disease) (Pony peach)	*Nanus mirabilis* Holmes Peach virus 4 Johnson *Prunivir mirabilis* Fawcett *Prunus* virus 3 (Hutchins) Smith Phony virus
Ring Spot (Ring spot complex) [Necrotic ring spot (?)] (See sour cherry yellows)	*Annulus cerasea* (Hildebrand) *Prunivir circummaculum* (Fawcett) Ring spot virus Necrotic ring spot virus (?)
Albino	Albino virus
Peach mosaic	*Marmor persicae* Holmes *Flavimacula persicae* (Holmes) McKinney Peach virus 6 (Johnson) *Prunus* virus 5 (Hutchins) Smith Peach mosaic virus *Prunivir flavimacula* Fawcett
Sour cherry yellows (Yellow leaf) (Physiological leaf drop) (Boarder tree) (Virus leaf drop) (See ring spot)	*Chlorogenus cerasae* (Hildebrand) Sour cherry yellows virus or viruses (Keitt and Clayton)
Prune dwarf (Prune mosaic) (Fellenberg mosaic) (Willows) (Shoestring of Italian prune)	*Nanus pruni* Holmes *Prunus* virus 6 (Thomas and Hildebrand) Smith
Red suture	*Chlorogenus persicae* var. *vulgaris* Holmes Red suture virus *Prunus* virus 4 (Bennett) Smith Peach virus 5 Johnson
Rosette mosaic	Rosette mosaic virus (Cation)
Muir peach dwarf (Muir rosette)	Muir peach dwarf virus
Wart (Blister)	*Galla verrucae* Blodgett *Prunivir verruca* (Fawcett)
Yellow leaf roll	Yellow leaf roll virus

Peach Yellows

History. Peach yellows undoubtedly is of American origin, since in the 150 years of its known history it has not been found in any other country. In the light of the discovery that the insect responsible for its dissemination is common on plum and further that the plum is subject to yellows, one may reason that the disease was indigenous on the wild plum and that in the early history of peach growing in eastern United States it was transferred from the plum to the peach. This speculation is further supported by the fact that the disease first appeared in a region where peaches and plums were extensively grown in close association for many years, as evidenced by the early horticultural writers.

Yellows as a disease of the peach is believed to have first appeared in the lower Delaware Valley. Peters in 1806 reported that a disease, which he called "yellows," occurred on his farm near Philadelphia as early as 1791. While his descriptions are not entirely accurate, it is evident from the history of the cases he mentions that he was dealing with true yellows. The fact that yellows was causing serious trouble at this time is evidenced by the fact that the American Philosophical Society in 1796 offered a prize of $60 "for the best method, verified by experiment, of preventing the premature decay of peach trees." It appears at the time of Peters' account of the disease that it was confined to a rather limited area, since the numerous statements of peachgrowers in other sections contain no reference to this trouble or any similar condition. Prince in 1828 gave a good description of the disease, in which the premature ripening of the fruit was first mentioned.

While it is possible that peach yellows may have had a multiple origin from the wild plums in different parts of the country, it seems more likely that it spread from the eastern area where it was first destructive to the West and North. It was reported from Michigan in 1866 and from Ontario, Canada, in 1878.

In 1888 Smith of the U.S. Department of Agriculture published the first of a series of reports on peach yellows. He gave a complete history of the disease to that date and carried out an extensive series of experiments by which he proved through a process of elimination that yellows must be due to a virus which could be transmitted only by a union of diseased with healthy tissues. He was not able to explain the origin of the disease in the absence of an actual union of tissues, but suspected that it might be transmitted by some insect. His experiments also indicated that seed from yellows-infected trees would not transmit the disease. Very little advance in our knowledge of the cause of peach yellows or the methods of transmission was made for a period of nearly forty years after the pioneer work of Smith. Experiments on different phases of the disease, involving the relation of nursery practices, cultural methods, and so forth, were extended by various investigators, but few new facts resulted from these. In 1916 Connors established the fact that the virus was not transmitted by the pollen. The first definite proof of insect transmission came in 1933, when Kunkel proved that the plum leaf hopper *Macropsis trimaculata* could transmit the yellows virus. This explained the spread of the disease in nature and gave a logical explanation of the geographic limitations of yellows. Kunkel also investigated a number of other possible insect vectors and concluded that these were not carriers of the virus. The results obtained by Kunkel were quickly confirmed and extended by Hartzell and by Manns. Hartzell investigated additional possible insect vectors and established more definitely the incubation period of the virus in its insect vector. He confirmed the results of other workers on nontransmission by pollen, seeds, or mechanical agents. Manns carried out extensive experiments on the manifestation of yellows in plum varieties and the distribution and abundance of the insect vector. He also investigated other possible carriers and established the fact that "little peach," another virus disease, was also transmitted by *M. trimaculata*. Kunkel in 1936 demonstrated by immunological methods that peach yellows and little peach are closely related, while peach rosette is distinct. He reported in the same year that yellows could be eliminated from an infected tree by subjecting it to high temperatures. The time of exposure and temperature required depended upon the tissues to be virus-inactivated; for example, the virus in diseased bud sticks held at 50°C (122°F) was inactivated in 3 to 4 minutes exposure, while young potted trees recently infected required 4 to 5 days at 95°F in a hot room, but young diseased trees in a dormant condition and free of earth were cured by immersing them for 10 minutes in water held at 50°C (122°F). Little peach and red suture also yielded to the heat treatment.

Geographic Distribution. Peach yellows is confined to the eastern United States and Canada. It does not occur in the southern part of this area and has never been reported west of the Mississippi River. In the western part of its range it is comparatively rare in Indiana, Illinois, and western Kentucky, but is rather prevalent in western Michigan. The first unquestionable case of the disease was found in Illinois in 1927, although earlier reports indicated that it might have been seen in this state before that date. The fact that it has not become established beyond the limit of its present distribution indicates that some limiting factor is definitely operative, since numerous opportunities for the introduction of the disease have occurred through the constant shipment of nursery trees into this area from the yellows-infested districts. The fact that peach yellows occurs farther south in the highlands than in the lowland eastern United States indicates that high summer temperatures may be an important factor in limiting its geographic distribution.

The insect vector is considered rare in Illinois, although it is occasionally found on plums.

Economic Importance. The loss from peach yellows is extremely severe in the regions where it is prevalent, since yellows trees never recover and are consequently a total loss to the grower. Epidemics of yellows seem to have occurred in certain periods, of which we have definite records for 1791, 1806–1807, 1817–1821, 1845–1858, 1874–1878, and 1886–1888. In these years losses were frequently so severe as to ruin completely many productive orchards. The life of an orchard is decidedly shortened when the disease becomes established. During quiescent periods 1 to 3 per cent annual loss of trees may be expected, while during epidemic periods this may be increased to 25 per cent. A loss of 3 per cent of the bearing trees in the regions where yellows commonly occurs would represent an annual killing of a half million trees and the consequent reduction of the crop. Since for a number of years eradication of diseased trees has been rigidly followed in the principal peach-growing areas, the loss is now much lower than during the past century.

Symptoms. Peach yellows may be easily confused with conditions of growth resulting from other agencies. For this reason an accurate diagnosis of the disease must depend upon a combination of symptoms or observations at the right season of the year. Under certain conditions "yellows" is a fairly descriptive name, but many other agencies may cause a yellowing of the foliage very similar to that resulting from the yellows virus, and consequently other symptoms are much more reliable in diagnosing the disease. Usually, the symptoms do not appear in the first year of growth after the tree is infected, although there are records of definite indication of the disease 42 days after inoculation by the leaf hopper. The following description is based on a well-developed case of the disease.

The *fruit* of yellows trees ripens *prematurely*, usually from 4 days to 3 weeks before normal maturity. It is not undersize and frequently is larger than normal. Reddish blotches or specks over the surface are evident in most cases. One of the most reliable symptoms is the presence in the flesh of reddish streaks and a decided deep-red color around the pit. The flavor of the yellows peaches is described as "insipid." Premature fruit may appear on one branch only, while the rest of the fruit on the tree may be normal.

The *tree* affected with yellows in an early stage is not markedly different from a normal tree, although it may be slightly off color as if suffering from malnutrition or winter injury, but on close examination certain characteristic symptoms may be seen. The yellows tree develops bunches of *slender, willowy shoots* which branch quickly, giving a "broom" effect to the whole growth (Fig. 61). The *leaves* developed on these shoots are narrow, yellowish, and red-spotted. The shoots have a tendency to grow vertically. In a normal shoot the primary growth is usually vertical, and the side branches grow outward, forming a wide angle with the main shoot. In yellows shoots the side branches tend to form a very narrow angle and quickly assume a vertical position. The willowlike growths commonly occur on the larger branches arising from adventitious buds, but they may develop in a terminal position anywhere on the tree. If they develop from the lower side of the branch, they curve upward much more rapidly than do normal shoots.

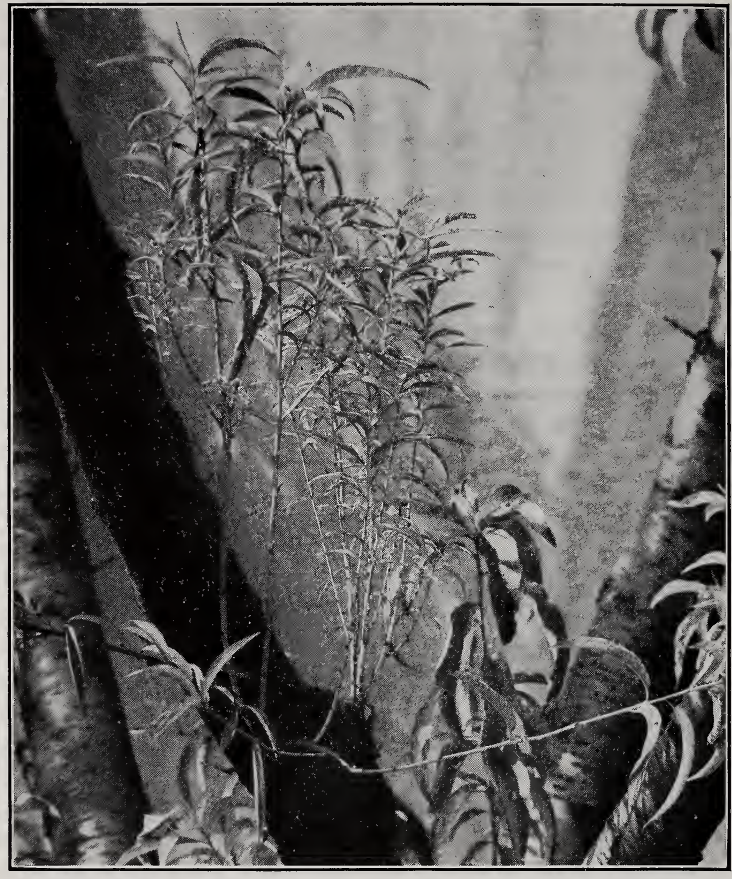

Fig. 61. Peach yellows, showing willowy, narrow-leaved sprouts arising from the tree trunk. (*Ill. Nat. Hist. Survey.*)

Affected trees set their buds earlier in the fall, and in the spring both the flower and the leaf buds develop earlier than in healthy trees. Sometimes diseased trees bloom in the fall, but abnormal growth conditions often cause this to occur on normal trees.

A tree which shows symptoms on only one branch becomes diseased throughout, although the other parts may continue normal development for a season or two. Soon the entire tree is weakened, the willowy growth occurs on many of the branches, and within 3 or 4 years the entire tree dies. After the second year the tree is of little value since most of the fruit ripens prematurely and is worthless.

The symptoms described above apply to fairly advanced stages of yellows. Early symptoms are more difficult to detect, but two of them may be mentioned. In diseased shoots the veins in the leaf are conspicuous owing to a "clearing" effect. This may be detected only when compared to normal leaves unless one is familiar with the condition. The second early symptom appears in very young leaves as they develop at the tip of the shoot. Such leaves when about ½ in. long on healthy shoots will stand up straight, but on diseased shoots they bend over and away from the main axis and the tips bend down, thus presenting a sicklelike appearance.

Abnormal growth conditions which might be mistaken for yellows may occur as a result of winter injury, borers, mice, or malnutrition. A general yellowing of the foliage together with a dying of the terminal twigs is characteristic of these injuries, but the slender water sprouts developed on such trees are not inclined to be bunched and the angle of the side shoots is not acute. The leaves of shoots on weak trees drop easily, while those on yellows trees are inclined to cling to the branch beyond the normal period of leaf fall.

Hosts. Until recently it was believed that peach yellows was confined to the peach (*Prunus persica*), nectarine (*P. persica* var. *nucipursica*), almond (*P. amygdalus*), and apricot (*P. armeniaca*). Manns demonstrated, however, that the yellows virus was present in certain of the Japanese and American species of plums and could be transferred to the peach either by budding or by the insect vector. In most varieties of plums the symptoms are decidedly masked and the trees may live for a number of years. Attempts to transmit the virus to cultivated cherries have been unsuccessful.

Causal Agent. Peach yellows is caused by a virus. The virus differs in many respects from those causing "mosaic" and similar diseases on many cultivated crops. A group of virus diseases is characterized by the fact that the extracted juices filtered or unfiltered are not infective and that the only known method of inoculation is by tissue union. The discovery of an insect vector for peach yellows and little peach has made this classification rather questionable.

The extensive experiments of Smith indicated that the disease was a specific one and was infectious. His work also seemed to eliminate such possible causes as malnutrition, winter injury, insect injury, bacteria, and fungi. Numerous experiments since this early work have proved that the virus can be transmitted from a diseased to a healthy tree by any process by which an actual union of tissues is brought about. These experiments have confirmed the fact that it is not transmitted by pollination; that seeds from diseased trees either fail to germinate or are free from the virus if they do grow; and that extracted juices inserted into the healthy plant will not transmit the virus.

One of the commonest methods of transmitting the virus is by budding. In nurseries, the diseased bud sticks may be taken either from bearing trees showing no marked symptoms at the time the buds were taken or from young trees in the nursery rows which had been inoculated the previous year, in which case the symptoms might not be in evidence. A high percentage of the trees in which buds from diseased trees are inserted will develop yellows in from 1 to 3 years. If the tissues unite but the bud does not develop a shoot, the seedling becomes infected. The virility of the virus in the buds seems to vary. Buds taken from willowy shoots are more virulent than those taken from a normal-appearing branch of a diseased tree. The minimum period of incubation in the field is about 1 year, but this may be shortened by forcing the inoculated trees in the greenhouse. On the other hand the first symptoms may not appear for 2 or 3 years after inoculation.

After the virus is introduced into a tree its progress through the tissue is rapid. The virus moves downward ten times more rapidly than upward. A tree inoculated some distance above the ground line will show symptoms much sooner than one budded at the ground line. Although the infected tree may show no symptoms for a period of several months, it has been demonstrated that the infective principle is present in distant parts of the plant within a few days after inoculation. All parts of the root system as well as the top are invaded by the virus.

The discovery of the insect transmission of the virus cleared up many obscure points in the development of the disease. The natural spread of yellows in the orchard after it had been established for several years could now be explained. The only insect known to be able to transmit the disease is the plum leaf hopper, *Macropsis trimaculata*, which feeds mainly on the plum but may be found also on the peach. It has been found in great abundance on both wild and cultivated plums. The plum leaf hopper according to Kunkel is found rather abundantly on the peach early in the season (May and June) but is rarely found in the later months when it seems to prefer the plum. It is also very shy and attempts to hide when disturbed. These habits may explain the fact that it was not suspected until recently as a possible vector.

The percentage of positive results from using this insect in transmission studies is low as compared to experiments with other virus diseases. For example, Kunkel secured seven positive cases out of 74 trees exposed. Hartzell secured an average of 16 per cent, but found that where the trees were exposed to the insects before June 22 he secured 36.4 per cent positive results. He was more successful in securing a higher percentage on trees of 3 to 5 years of age than on 1-year-old trees. In his experiments he found that the time required to produce the first symptoms varied from 42 to 268 days and that the maximum incubation period of the virus in the insect varied from 10 to 26 days with a minimum of 7 to 8 days.

The fact that yellows in a masked form is common on plums and that the plum is a favorite host of the leaf hopper leads to the conclusion that many cases of yellows in peach orchards result from the presence of wild or cultivated plums in the neighborhood. The restricted distribution of yellows and the epidemics which have occurred in the past may also be explained as a result of the discovery of the insect vector. Manns has found that the distribution of yellows coincides rather closely with the known distribution of the plum leaf hopper.

Control. During the period when it was thought that yellows might be due to nutritional disturbances, numerous experiments on the use of fertilizers were undertaken and reported in detail. From these experiments it seems certain that the virus is not affected by soil conditions. Spraying also failed to control the disease, although in the light of the discovery of the insect vector, some control might be expected where sprays are applied which might repel this insect.

No variety of peach, apricot, or nectarine is known to be highly resistant to the disease, and it is now known that plums which have been used in some regions as rootstocks for the peach are also susceptible.

Marked success has been secured by frequent state-wide inspections and destruction of yellows trees as soon as the first symptoms can be detected. The results obtained in Pennsylvania over a 7-year period of careful inspection and eradication will illustrate this point. In 1921 the inspectors found 4.45 per cent yellows trees in the orchards. In 1927 the cases had fallen to 0.23 per cent. This decrease was very marked after the first 3 years of eradication, as would be expected in view of the fact that the symptoms of trees infected during the first year or two of inspection would not be evident until a year or so later and the disease could be transmitted to healthy trees during this period.

Eradication of plums in the neighborhood of peach orchards should aid in the control of yellows, since the plum is a favorite host of the plum leaf hopper which transmits the disease. Wild plum thickets are common throughout the entire region where yellows occurs, and the symptoms of the disease are often masked in the plum. Growers should avoid growing plums either as interplants or in close proximity to peach orchards.

At one time it was thought that most of the yellows cases originated in the nurseries, and these were blamed for the dissemination of the disease. Extensive data secured by McCubbin (1925) and others indicate that very few cases originate in the nurseries. Nevertheless, the nurseryman should be required to secure budwood from trees which, by careful inspection, do not show symptoms of the disease. Kunkel has demonstrated that the yellows virus can be inactivated in bud sticks by immersion in hot water held at 122°F (50°C) for a period of 5 minutes. Since this treatment does not injure the buds, it would be practical for the nurseryman, in the case of doubtful budwood, to immerse the bud sticks in a hot bath for 5 minutes. Trees budded and held in the nursery row, if suspected, can be cured by immersion during dormancy in a tank of water held at 122°F for a period of 10 minutes. In view of the limited number of cases of yellows arising from nursery practices, it is doubtful if the heat treatment is necessary if other precautions are taken, except in cases where extreme measures are called for, such as might occur when valuable stock is known to have been exposed to infection.

Kunkel (1936) has demonstrated that peach trees infected with yellows may be immunized by inoculating them with the little peach virus and that little peach–infected trees may be immunized by inoculating them with the yellows virus. But in each case the trees developed the symptoms produced by the immunizing virus. Since both of these diseases are destructive, this method would be of no practical value unless an attenuated virus strain of one or the other of the diseases could be secured. In case attenuated strains are secured, the simple insertion of a bud from trees previously inoculated with these strains on a healthy tree would immunize it against either disease.

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Little Peach

History and Distribution. At present little peach outranks yellows as the most important virus disease of the northern peach-growing areas. It was first described by Taft from Michigan in 1896. By 1925, it was found to be generally distributed over the northeastern states and southeastern Canada. It has about the same geographical distribution as peach yellows.

Symptoms. In some respects the symptoms of little peach are similar to those of yellows, to which it seems closely related. As the name implies, the *fruit* is reduced in size and ripens several days to 3 weeks later than normal fruit. There is no evidence of the splotched appearance, as in yellows. The kernels in the pits are undeveloped, or if present, fail to germinate.

The first symptoms on the tree are usually conspicuous on only one or two main branches. The foliage at first is darker green and more compact than normal. More buds than normal develop along the main branches, but the willowy growth characteristic of yellows is lacking, and the vertical growth of the laterals is not so pronounced. The shoots are somewhat shortened and appear bushy, not so much from shortening of internodes as from the development of more laterals, thus increasing the number of leaf clusters. The *leaves*, at first dark green and leathery in texture, droop and bend inward toward the shoot. Later they may turn slightly yellow, but do not have the characteristic purple spotting of yellows.

Causal Agent. The virus has been given the following names: *Chlorogenus persicae* var. *micropersica* by Holmes, *Prunus* virus 1A by Smith, and peach virus 3 by Johnson.

Hosts. Little peach has approximately the same host range as peach yellows. Several plum species are symptomless carriers: for example, Myrobalan plum (*Prunus cerasifera*), which is extensively used as rootstock, shows no evidence of the disease but will transmit it to varieties grafted or budded on it.

Immunological Relationships. Kunkel has demonstrated that trees having little peach virus are immune from infection by the peach yellows virus and vice versa. He also showed that when peach seedlings are simultaneously inoculated with the two viruses, the bud placed in the top position determines the nature of the acquired disease. In the orchard some trees may occasionally show evidence of both diseases, little peach on one branch and yellows on another, so there is some question about the effective immunization of older trees.

Heat treatment shows the same inactivation point as for peach yellows, and the fact that little peach virus will immunize against peach yellows has led some to regard these two diseases as being due to strains of the same virus. Furthermore, the virus is spread by the same vector, plum leaf hopper, has a similar host range, and has approximately the same geographical distribution.

Control. Removal of diseased trees and of wild hosts which may be masked carriers are the only control measures necessary, but spraying for control of the plum leaf hopper may aid.

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Peach Rosette

History and Distribution. The first accurate description of peach rosette was given by Smith in 1891, although it had been known for at least 10 years previous to this date and was referred to earlier by Smith as probably a southern form of peach yellows, since it was found in Georgia and had not been observed in the northern peach area where the typical peach yellows was common. Smith proved that the disease could be transmitted by budding and other methods of tissue transplanting and also recorded the fact that a similar, if not identical, disease was found on plum. McClintock (1923) found that peach rosette could be transmitted by budding to the sand cherry (*Prunus pumila*), as well as to the peach and plum, and Thornberry (1941) transmitted peach rosette from apricot to peach.

Since the early work of Smith, the distribution of the disease has been extended to include the states of Arkansas, Kansas, Illinois, Oklahoma, West Virginia, Kentucky, Tennessee, Mississippi, South Carolina, and Georgia. While it is regarded as a disease of the southern border of the peach-growing area, it has been observed by the writer as far north as East St. Louis in Illinois (1928). A similar disease has been reported from Italy by Farraris (1928).

Economic Importance. While it is true that trees affected with rosette are killed much more quickly than those having any of the other virus diseases, it has been demonstrated that, over a period of years, yellows is more destructive than is rosette. Sporadic outbreaks of severe rosette have occurred when it appeared that it would become extremely destructive, but the expected extensive spread of the disease has not occurred. This may be due in part to the fact that unlike the other peach virus diseases, peach rosette kills the trees within a year, and consequently, unless conditions are very favorable, little opportunity is afforded for its dissemination. Further, after the symptoms become evident, there is little chance of nurserymen selecting buds from such trees. Trees in an orchard which show the striking symptoms of rosette are likely to be destroyed at once by the grower, thus further reducing the chance of dissemination.

Symptoms. Symptoms of rosette may vary according to the conditions under which they are inoculated. Young, active trees, if inoculated in the spring or early summer some distance above the ground level, may wilt suddenly and die, with no evidence of the characteristic symptoms described below. Older trees, even when inoculated in the top, are not likely to die until later. Young trees inoculated at the ground line or below also show characteristic foliar and twig symptoms.

Ordinarily the first symptoms appear in the early spring when the buds first open. Only a few of the dormant buds on a healthy tree develop in the spring, while on one having rosette, most of these buds will start growth immediately. Buds on the larger branches, which normally would remain dormant, also open as though starting new shoots. In a healthy tree the few shoots quickly extend into a growth of a foot or more, while in the rosetted trees the new shoot attains a length of only 1 to 3 inches. In a normal shoot most of the buds developed in the axils of the leaves on the terminal part of the shoot remain dormant until the following year, but in rosette these buds start a new growth immediately. In other words, the rosetted trees continue in an active condition with all processes of growth speeded up but with no chance for maturing and normal elongation of parts. The laterals from the short, primary growth never grow more than an inch or so in length, and the few buds formed on these immediately produce leaves.

The *leaves* on a rosetted branch are multiplied enormously by the above process and appear in compact tufts of from 200 to 400 small diminutive leaves, thus giving rise to the common name of the disease (Fig. 62). The leaves are normal green at first or even darker than those on a healthy tree. The basal leaves on a rosetted branch may be fairly large, but with a pronounced inrolling of the margin. Unlike yellows, the leaves on a rosetted tree are easily detached and are dislodged by vigorous shaking. The younger leaves remain green, and the red blotching characteristic of yellows does not appear.

The *fruit*, if formed at all, does not ripen prematurely. Ordinarily, the fruit falls from the tree before reaching maturity.

On old trees in which one or two limbs only may show the disease, the tufted terminal growth may be seen at a distance. Trees showing a trace of rosette in one season are likely to be completely diseased the following year and will die before the end of the growing season.

Causal Agent. It has been proved by many investigators that peach rosette virus can be transmitted by budding. In nature, the disease seems to spread rather erratically. In a farm of 100 acres of peaches, the disease appeared in 1887 on two trees and had increased to only 47 by 1891. Other records show a much more rapid spread.

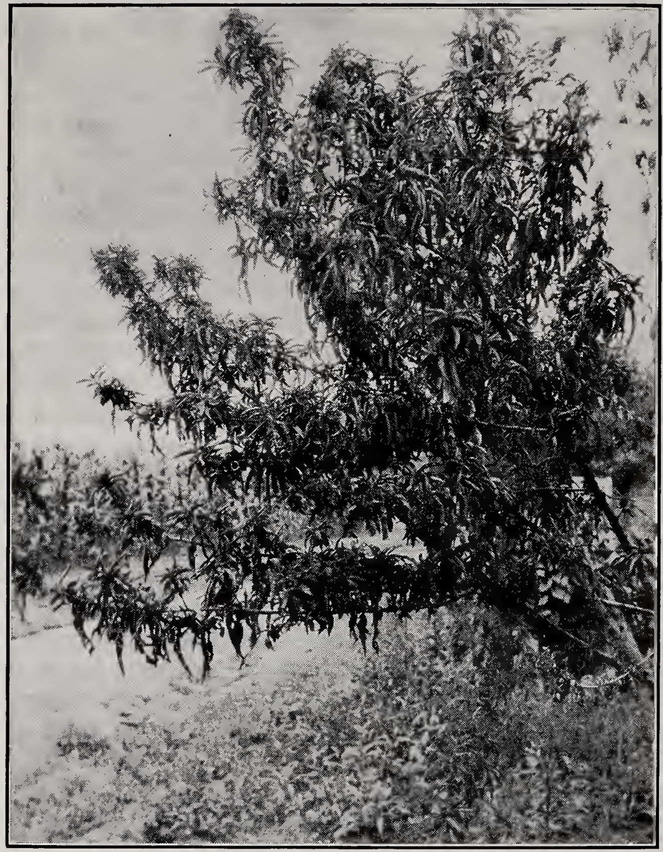

Fig. 62. Peach rosette showing bunched leaves.

While it seems evident that some insect must be responsible for the dissemination of the virus in nature, the only proved method of transmission has been by live-tissue union. If the tissue fails to unite, the virus is not transmitted. McClintock (1923, 1931) and others report that a number of insects commonly found on the peach and plum failed to transmit the virus.

Kunkel has demonstrated that the virus of peach rosette is not closely related to that of either peach yellows or little peach, since trees infected with these diseases, when inoculated with rosette virus, showed typical rosette symptoms in about 5 weeks after they were budded. Kunkel also showed that, while the rosette virus can be inactivated by heat, it is more resistant than is the yellows or little peach virus.

Hosts. Peach rosette occurs commonly on the peach and plum. It has been transmitted to the Chickasaw plum (*Prunus angustifolia*), Red June plum (*P. salicina*), Maynard plum (*P. salacina × simonii*), almond (*P. communis*), apricot (*P. armeniaca*), sour cherry (*P. cerasus*), sand cherry (*P. pumila*), and Mazzard cherry (*P. avium*). The Marianna plum probably is immune.

Control. The same general program as that used for other virus diseases of the peach should be followed, with special emphasis on the eradication of wild hosts on which the disease is known to occur.

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X-diseases of Peach and Cherry

A group of peach and cherry virus diseases which seem to be closely related but show distinct characteristics on some hosts and under different climatic conditions are here treated under the collective term "X-diseases." On peach and on chokecherries (*Prunus virginiana* and *P. virginiana* var. *dimissa*), the X-disease of the eastern United States and Canada has very similar symptoms to the western X-disease. On the other hand, several virus diseases of both sweet and sour cherries, when cross-inoculated onto peach, all produce symptoms similar to typical X-disease, although the symptoms on cherries may be quite varied. This variation on cherries may be due either to different strains of the same virus or to such variables as climate or rootstock on which the cherries are grown. It is also possible that, in some cases, an unknown virus is present, and thus the symptom expression may be the result of the combined action of the two or more viruses.

On cherries one symptom seems to be common to all of these diseases. This consists in the presence of undersized and off-colored fruit and a general decline in production. For this reason the name "little cherry" is often applied to these diseases. Another name, "buckskin," is used to describe a very important disease of sweet cherries in California, but here again the fruit is reduced in size and is off color.

The composite treatment of these diseases which follows does not imply that one distinct virus is responsible for all of the diseases included, but that the similarity of symptoms together with some recent cross-inoculation results justifies such a treatment. Included in this grouping are "buckskin" of cherry, "little cherry," "small bitter cherry," "western X-disease" of peach and cherry, "eastern X-disease" of peach, and "peach leaf casting yellows."

In 1931, Rawlins and Horne described a destructive graft-infectious disease of cherries which had been observed as early as 1928 in a number of orchards in northern California. Fruits on diseased trees were dwarfed, conical, and at the blossom end developed a dull, off-color area from which the name "buckskin" was derived. Later it was proved that a disease of peach called "leaf casting yellows" was caused by this same virus. By 1950, it was generally agreed that the western X-disease and possibly the eastern X-disease were caused by strains of the buckskin virus.

While the "little cherry" diseases were being investigated in the West, a new peach disease was found by Stoddard in Connecticut. He reported on this disease in 1933 and in a year or so later gave it the name X-disease. The relation of this new disease to the premature coloration and rosetting of eastern chokecherries was established by graft inoculations. As early as 1936, Richards recorded abnormal reddening of chokecherry leaves in northern Washington, which was subsequently identified as the western X-disease. This was regarded as differing in some respects from the eastern form.

Economic Importance. If one accepts the broad conception regarding the host range of this virus, it becomes one of the most important virus diseases of both cherry and peach. Many sweet cherry orchards in California have become so unproductive as a result of the disease that they have been removed. In Oregon, Washington, Idaho, and British Columbia, surveys have revealed a high percentage of infected trees, both sweet and sour cherries as well as peach. If the eastern X-disease is considered as a strain of the buckskin virus, the loss would include the extensive destruction of peach in eastern sections of the United States and Canada.

Geographic Distribution. Buckskin of cherry, leaf casting yellows of peach, and western X-disease are widely distributed in British Columbia, Washington, Oregon, Idaho, Utah, and California and have been reported in Colorado. On chokecherry either the western or eastern X-disease extends across the entire northern United States and southern Canada wherever this wild cherry (*Prunus virginiana* and *P. virginiana* var. *dismissa*) occurs. On peach, the eastern X-disease is most prevalent in Connecticut, Massachusetts, and New York and has been reported on this host in New Hampshire, Ontario, Canada, Ohio, Pennsylvania, and Illinois. In New York eastern X-disease is important on cherry.

Symptoms. *Cherries.* Symptom expression on both fruit and tree varies with the varieties, rootstock used, and other little-understood factors. On the *fruit*, the most common evident symptom is dwarfing, aptly called "little cherry." The fruit on sweet cherries on Mazzard rootstock gives the most useful diagnostic symptoms. Fruits fail to mature, remain small, are conical in form, hang on the tree after normal cherries have dropped, and fail to develop the normal color of the variety concerned. The pedicels are short, and the apex of the fruit shows a dull area. Cherry trees on Mahaleb show wilt and decline. One virus strain (Napa) differs from the above in that the fruit, while undersized, drops at the normal ripening time and retains the luster of normal fruit. Diseased trees appear normal during most of the growing season, but in the late summer and early fall the *leaves* show an orange-red color along the basal portion of the midrib. Retarded growth and die back occur in older infected trees.

On *peach*, the symptom expression of the virus is much the same for eastern and western X-disease and leaf casting yellows. Variations may be due to strains of the virus or to conditions under which the host plants are growing. The eastern X-disease symptoms on peach will be described here, and any deviation from these symptoms for the western form and leaf casting yellows will be noted. Peach trees appear normal in the spring for some 6 or 7 weeks after growth starts. After this period the foliage shows a diffused blended yellow and red discoloration with a longitudinal upward curling of the leaf edges. The discolored areas are irregular in shape and vary in size. The areas outside the colored spots become chlorotic. The leaves become brittle, and the discolored spots fall out, giving a tattered appearance to the leaf blade. Starting about mid-season, defoliation begins, and by late summer all the leaves except a few at the tips of the twigs are lost.

For the most part the fruit on affected trees shrivels and drops, and any remaining ripens prematurely. Seeds in the pits do not develop. Bearing trees in the orchard, especially the older ones, do not die, but are weakened to such an extent that they are easily killed by low temperatures and are entirely unproductive.

The symptoms on *chokecherries* as described by various workers appear to be much the same for both eastern and western X-disease, as well as cherry buckskin, except for the apparent absence of the rosette effect on the western chokecherry.

The most striking symptom on the chokecherry is the appearance of the red and yellow foliage in late summer before there is any evidence of normal fall coloration. On both the eastern and western chokecherry this starts as a yellow coloration by late June and reaches the orange and deep-red stage in late July and August. In the second year after infection, there is a decided stunting of the shoots with a rosetted effect resulting from the shortened internodes at the tip. Frequently, many of the diseased chokecherries die out in the third or fourth year, but a few may remain in a stunted condition for a longer period.

Hosts. Considered as a composite virus, the known host range of the X-diseases is very extensive. In many cases the only proof of susceptibility has been by cross inoculation. Symptoms are produced on all varieties of peach (*Prunus persica*), on nectarine, almond, wild goose plum (*P. munsoniana*), Mahaleb cherry, and on a number of varieties of sweet and sour cherries. Wild hosts include the eastern and western chokecherry. There is still some question about the susceptibility of the apricot. By the use of dodder, Kunkel was able to transmit the eastern X-disease virus to several herbaceous plants, including tomato, carrot, parsley, and periwinkle. In 1954 Gilmer, Moore, and Keitt reported transmission of the X-virus to 15 species of *Prunus* not previously reported and confirmed previous reports of transmission to *P. americana*, *P. avium*, *P. bessyi*, *P. cerasus*, and *P. japonica*.

Transmission. The natural spread of the X-diseases indicates insect vectors. Western X-disease virus was successfully transmitted in 1950 by the geminate leaf hopper (*Colladonus geminatus*). Gilmer in 1954 reported successful transmission of the eastern X-disease virus by *Colladonus clitellarius* (Say.). Thornberry (1954) reported possible transmission by this insect in Illinois. In the East, natural spread is always from chokecherry to peach; rarely, if ever, from peach to peach or from peach to chokecherry. On the other hand, the western X-disease spreads from peach to peach, and the chokecherry may not be involved. These differences may be due to the presence or absence of certain insect vectors, lack of other food plants for the insects, or to differences in strains of the virus.

Control. In the eastern United States, the X-disease is easily controlled by the elimination of chokecherries within 500 ft of peach trees, since the disease does not spread from peach to peach. In the West, where the western X-disease spreads from peach to peach, this method must be supplemented by roguing the diseased peach trees. Early recognition of the disease and prompt removal of affected trees, together with the elimination of chokecherries, have solved the problem of control in most areas. Most fruit-growing states have an inspection service which aids the fruitgrowers in locating any trees which might harbor the virus.

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Phony Peach

History. According to Hutchins et al. (1951), this virus disease, also called "pony peach," was recognized as early as 1885 by Samuel Rumph, who described a "pony," or dwarf type, of Elberta. At first the possibility of its spread to normal trees was not recognized. By 1915 growers in central Georgia realized that this was really a communicable disease, since by that date thousands of trees were affected. Research into the cause of the disease was started by the U.S. Department of Agriculture in 1921. While it was suspected that the malady might be due to a virus, the difficulty of transmission by ordinary techniques delayed positive proof until about 1928. Hutchins found that successful transmission could be accomplished only by grafting the roots of a diseased tree to a healthy one or by use of sections of whole roots, thus establishing the fact that the virus was in the wood rather than the bark of the plant. In the meantime the disease had spread over a large section of central Georgia and into eastern Alabama. A Federal quarantine was established in 1929 which served to restrict the movement of nursery stock within and from affected districts. Later state and Federal agencies cooperated to survey and eradicate the diseased trees from all the states where it was found. This proved to be especially effective in those states where only a few diseased trees were discovered.

Economic Importance. Phony peach ranks first in importance in the southeastern section of the United States. As a result of the efficient eradication program, it is of little importance outside of this area. Over a million and a half trees have been eradicated since 1929, and prior to that time it is estimated that over one million trees had become worthless in the central Georgia region as a result of phony peach.

The main loss in affected trees is the gradual but marked reduction in production, although the diseased trees continue to thrive for a period of years following infection. Trees that become diseased before they come into bearing never produce a profitable crop. Bearing trees are worthless 3 or 4 years after infection because of off-grade fruit and reduced production.

Geographic Distribution. As a result of the extensive surveys from 1920 to 1947, phony peach was located in all the Atlantic Coast states below Virginia and in all the Gulf states. Scattered infected trees were found in bordering states, including Oklahoma, Missouri, Kentucky, Tennessee, Maryland, Pennsylvania, and in southern sections of Illinois and Indiana. It has never been reported from abroad or from the western or northern peach-growing areas in the United States.

Hosts. All varieties of peach are known to be susceptible to natural infection as well as seedlings of apricot, Japanese apricot (*P. mume*), chickasaw plum (*P. angustifolia*), and Mexican plum (*P. mexicana*). Artificial inoculations by use of piece-root grafts from infected trees show that other species of *Prunus* such as almond (*P. amygdalus*), David peach (*P. davidiana*), Hortulan plum (*P. hortulana*) are susceptible. So far as now known the virus is limited to the genus *Prunus*.

Symptoms. When in full foliage a "phony" peach tree is not strikingly different from the surrounding healthy trees. The whole tree has a compact appearance due to the shortened internodes of the terminal and lateral shoots and dense dark green foliage. The outline of the crown is sharp with no upright shoots extending beyond the dense growth. The diseased trees leaf out earlier than normal ones and hold their foliage longer in the fall.

Phony trees bloom earlier than normal, and the fruit produced ripens a few days early. The fruits are reduced in size and in number but are more highly colored than normal. Seeds from phony trees germinate well and produce normal seedlings.

Since there is no yellowing, rosetting, or other abnormal leaf symptoms, the only method of detecting phony peaches in the orchard is by the character of growth of the entire tree. Fortunately, a relatively simple chemical identification test was devised which could be applied either in the laboratory or in the field. This consisted of acidulated absolute methanol (100 cc methanol, to which is added 15 to 20 drops of chemically pure concentrated hydrochloric acid). Thin sections of whole roots immersed for a few minutes in this solution will show purplish spots scattered through the wood cylinder, while normal root sections will be free from color or show only a faint, even, purple tint. If left too long, the normal root sections as well as the phony may develop a uniform purple color.

Transmission, Natural Spread, and Incubation. Great difficulty was experienced in determining the method by which this virus could be transmitted. Peach virus diseases such as yellows and rosette were easily transmitted by budding or grafting long before insect vectors were found. Attempts to transmit phony peach virus by these methods at first were entirely unsuccessful. Finally, it was discovered that grafting whole root pieces from diseased trees on roots of normal trees would transmit the virus. It seemed, therefore, that the virus was confined to the woody cylinder of the roots, especially since top grafting of diseased branches on healthy limbs failed to transmit the disease. Later it was established that phony virus occasionally exists in the tops of affected peach and plum trees. It is evident, however, that the virus is contained mainly in the woody cylinder of the roots, and the aboveground effect is due to the interference of normal root functions by the virus.

Except in concentrated centers of infection the natural spread of the disease is very slow. The method of the natural spread remained a mystery for a number of years. Since the virus seemed to be confined to the roots, it was natural to suppose that root-feeding insects were responsible for transmission. Efforts to locate such vectors were unsuccessful. In 1949, Turner reported the successful transmission of phony peach virus by four species of leaf hoppers (Cicadellidae). These insects limit their feeding to small branches of the peach, thus indicating that the virus at times may be present in these organs.

The length of the incubation period under natural conditions is not known. When healthy trees are planted in close association with diseased trees, 3 to 5 per cent show symptoms during the third growing season. When nursery trees are inoculated by root-piece grafts, the incubation period is about 18 months. When healthy cions are grafted in the tops of phony-diseased trees, the growth from the cions shows typical symptoms at once. A dormant healthy nursery tree grafted by the taproot to roots of a phony tree will show symptoms immediately after growth starts.

Control. Eradication of the scattered phony peach trees in borderline states has solved the problem of control, but in centers of heavy incidence of the disease, eradication has been more difficult. At present, however, roguing is the only effective method of keeping the disease in check.

Wild plum and peach seedlings in the neighborhood of commercial peach orchards should be destroyed. When such trees have the virus infection, it is difficult to detect, and, furthermore, the virus may be present and transmissible for 2 or 3 years before any symptoms appear.

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The Ring Spot Complex

While usually of no great economic importance so far as reduced production is concerned, ring spot is so widely distributed on stone fruits and so complex in its relation to other viruses that it cannot be ignored in a treatment of stone fruit virus diseases. Apparently the ring spot virus is a complex of many forms or strains with widely varied manifestations when in different hosts or under varying environments. When in association with other viruses, the situation becomes even more complex.

Included in the ring spot virus complex are several diseases of peach, cherry, and plum, which vary mainly in symptom expression. These include necrotic ring spot and tatter leaf.

Of late years it has become apparent that in one form or another this virus is generally distributed throughout the United States and Canada and is probably prevalent in European and Asiatic countries. In many cases the presence of the virus in a given plant can be determined only by transmission to a symptom-expressing host and under conditions known to give visible symptoms. It is known to exist in most of the stone fruits, both commercial and wild. It is probably most widespread in cherries, and that cherries serve as reservoirs of the virus is suggested, since in regions where cherries are not commonly grown the disease is less prevalent on other stone fruits. To what extent it occurs in hosts other than stone fruits is not known, except that it has been recovered from rose.

Symptoms vary, owing to differential resistance of the hosts and to the existence of forms of the virus. Symptoms on the leaves consist of chlorotic or necrotic patterns made up of rings and spots. The size of the rings is inversely proportional to the number present. Symptoms are more evident in early stages of the disease, and in many hosts visual leaf symptoms occur only during the first year of infection, although it can be demonstrated that the virus continues to be present in subsequent years. On peach varieties having red-colored fruit the rings are bordered with red. Some susceptible varieties of peach are retarded in their growth, and terminal die back may result. Plums for the most part show mild symptoms with little injury either in production or tree growth.

On sweet cherry, symptoms when present vary from chlorotic ring patterns to necrotic spotting. On sour cherries the symptoms are confusing in that the ring spot pattern often occurs only during the first growing season and is often absent in subsequent years.

A severe form of ring spot occurs on Japanese flowering cherries, especially on Shirofugen. When inoculated with diseased buds the virus moves slowly into the surrounding tissue. The invaded tissue becomes necrotic, and the whole plant eventually will be killed. For this reason the Shirofugen cherry is an excellent indexing host.

One interesting fact in regard to this disease is that it may be seedborne. Cation found upward of 10 per cent of Mahaleb cherry seedlings grown from commercial seed developed the ring spot disease. This is unusual in the transmission of stone fruit viruses.

The agents responsible for natural spread of the disease are not known.

Since ring spot is so generally distributed and of doubtful economic importance, no great effort has been made to control it. Virus-free foundation stock may be of use in those cases where the disease is of importance from the standpoint of production or where experimental work is undertaken on the complex relationship of this virus to other viruses.

Another virus of the ring spot type, *necrotic leaf spot,* is said to cause injury to June-budded nursery stock of peach in California. The symptoms, necrotic spotting and shot hole, are present only in the current season, the disease being symptomless in subsequent years. [H. K. Wagnon and J. R. Breece, *Calif. State Dept. Agr. Bull.* **3**:13–17 (1954).]

Albino

This is primarily a sweet cherry disease which at present is limited to the Rogue River Valley in southern Oregon where it was discovered in 1937. In this region it is of great economic importance since it has been found to spread rapidly and soon kills the affected trees. If allowed to spread to other cherry-growing areas, it might become the most important of the cherry virus diseases. In nature the disease has been reported only on sweet cherry, but has been experimentally transmitted to Montmorency sour cherry.

Symptoms and Transmission. All varieties of sweet cherries show about the same symptoms. Die back of diseased branches begins in early spring as soon as leaves appear. The leaves become golden bronze with up-rolled margins. In late summer new growth of small, green, rosetted leaves is produced from terminal buds. Fruits remain small and turn white. On experimentally inoculated trees all those on Mahaleb rootstock, with the exception of Black Tartarian and Montmorency (sour), were killed by the albino virus during the summer after inoculation. When on Mazzard many of the trees lived for as much as 3 years, in spite of a severe shock reaction the first year. These trees were dwarfed and rosetted with light foliage, and the fruit when produced was typical of that found in naturally infected trees. The only known method of transmission is by tissue union.

Peach trees inoculated from diseased cherry showed no symptoms, and back inoculation to sweet cherry indicated that peach does not carry the virus. Whether the virus is latent in other species of *Prunus* has not been determined.

Relation to Other Cherry Virus Diseases. Many of the symptoms of albino are similar to those of buckskin (see X-diseases of peach and cherry); however, the uniform golden-bronze leaves associated with albino are not listed as a symptom of buckskin. The western X-disease associated with buckskin of sweet cherries, which is readily transmitted to peach, differs in that the albino virus when inoculated into peach does not develop any symptoms such as result from the western X-disease. Furthermore, peach trees interplanted with cherries in the section where albino occurs have not developed any symptoms of a virus disease.

Control measures have not been attempted in connection with this disease, but since it may be spread to other important cherry-growing areas, nurserymen are warned to avoid propagating material from this region. Since symptoms appear shortly after inoculation, an eradication program would seem feasible if carried out on a regional basis. [S. M. Zeller, J. A. Milbrath, and C. B. Cordy, Albino cherry, a virus disease in Southern Oregon. (Abstr.), *Phytopathology,* **34**: 937 (1944).]

Peach Mosaic

History and Geographic Distribution. Peach mosaic was not discovered until 1931, although it was probably present in southern California and Texas some years before this date. Nothing is known of the origin of the disease, but the fact that it occurs on a number of species of plums without distinct symptom expression and that these have been extensively planted in the southwestern region of the United States along ditch banks and irrigation canals indicates that the virus may have been present in these hosts and was transmitted to peach orchards when these were planted in the area. In 1931, the disease was observed by Hutchins at Bango, Texas, and by Bodine near Palisade, Colorado, and in 1932 Hutchins described it as a new virus disease to which he gave the name "peach mosaic." Extensive state and Federal surveys between this date and 1940 revealed its presence in southern California, Texas, Arizona, New Mexico, southern Oklahoma, western Arkansas, western Colorado, and in Mexico. Transmission experiments by Cochran and Hutchins extended the range of hosts to numerous varieties of peach, apricot, and plum.

Economic Importance. Peach mosaic is limited in its geographical distribution to the southwestern United States, but the region contains extensive areas of commercial peach orchards, especially in southern California, so that reduced production as a result of the disease is of considerable economic importance. For example, in 1936 out of a half million trees inspected in southern California over 28,000 were found affected. In 1937, three-quarters of a million trees inspected revealed 34,239 with mosaic.

The effect of the virus on susceptible varieties in reduction in quality and quantity of the fruit is such as to render them unprofitable. On a number of clingstone varieties, which as a class are tolerant to the disease, no serious loss occurs. When these are grown in the neighborhood of the susceptible freestones, the hazard of transmission must be considered.

Host Range. Mosaic is limited to the genus *Prunus* so far as now known. Within this genus the cherry group seems to be immune. Peach, apricot, nectarine, and almond and domestica, Japanese, damson, chickasaw, wild goose (*P. munsoniana*), Myrobalan, and Mexican plums have been experimentally infected. The virus has been recovered from many naturally infected horticultural varieties of peach and apricot and from a few varieties of Japanese plums. It has also been recovered from a number of naturally infected plums. Chickasaw plum (*P. angustifolia*) is important in that it is a symptomless carrier, and many infected trees have been found in the extensive thickets of this species in the neighborhood of mosaic-infected peach orchards in Texas.

Symptoms. Apparently there are a number of strains of the peach mosaic virus, and the symptom expression may vary according to the strain involved. Furthermore, great variation occurs in the different varieties and species of *Prunus*.

On a very susceptible variety such as J. H. Hale a severe form of the virus will show the following symptoms. In the first year of infection the early-formed leaves may wither and fall or may be irregular in shape with yellow and green patterns of markings. Some veinlet clearing may be evident. Dwarfing as a result of shortened growth of the twigs is pronounced after the first year of infection. By the second year diseased trees apparently go into a chronic stage of the disease, with well-developed mottle and reduced shoot growth but with less defoliation. Bumpy fruit is characteristic of severe types of infection on some varieties. Some virus forms do not affect the fruit.

Varieties having large pink flowers show a striking symptom. This consists of a variegated pattern on the petals consisting of streaking and boat-shaped spots. This "breaking," as it is called, is common in some virus-infected flowers such as the tulip, but seems to be rare in fruit diseases. It is not a symptom in any other virus disease of stone fruit except in peach ring spot. It is not evident on mosaic-infected trees of the small-flowered types such as Elberta and J. H. Hale.

Symptom expression on hosts other than peach varies with the form of virus involved. On apricot, almond, and various plum species, the peach mosaic virus produces brilliant patterns of mottling of the ring spot, splotchy, irregular, or oak leaf types. However, there is no correlation between the symptom type on these hosts and that on peach. A brilliant mottling on apricot may result in no such symptoms when this virus strain is transmitted to peach.

Some plum varieties may carry the virus without showing any symptoms. This increases the problem of control where such varieties are grown in the neighborhood of commercial peach or plum orchards.

Transmission and Incubation Period. *Eriophyes* sp. mite has recently been shown to be a vector of this disease. [Wilson, N. S., L. S. Jones, and L. C. Cochran, *Plant Disease Reptr.*, **39**: 889–892 (1955).] The rate of spread in nature varies with the locality and the number of diseased trees present. Artificial transmission of the virus has been effected by tissue grafting from peach to peach by the use of fruit, leaf, stem wood, root wood, and bark. If inoculations are made by cions at the time of breaking of dormancy, symptoms may appear as early as 14 days thereafter. If grafts are made when the leaves are partially expanded, the incubation period may be as long as 6 weeks, and if made as late as June, symptoms usually do not appear until the following season.

Control. Federal and state eradication programs in the commercial peach-growing areas together with restrictions on the movement of nursery trees and cion wood have successfully reduced the peach mosaic problem in most of these areas. Careful attention in the selection of sites for new orchards and selection of varieties known to show little damage from virus infection is advised in borderline areas.

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Sour Cherry Yellows

This most important virus disease of sour cherries in the United States and Canada is of interest not only because of the economic loss but also because of the complex nature of the viruses concerned. The extensive research on the disease clearly indicates that typical symptom expression is the result of the combined action of two or more viruses.

Other common names for the disease are yellow leaf, physiological leaf drop, physiological yellows, boarder trees, and virus leaf drop.

Economic Importance. The main loss from cherry yellows is the reduction in yield, which may amount to as much as 50 per cent, 2 to 5 years after the first symptoms appear. The rate of spread within an orchard as determined by surveys in Wisconsin averages approximately 3 per cent annually, but varies greatly from year to year. Old orchards may have practically all the trees affected. The life of the diseased trees is not materially shortened.

Geographic Distribution. In the United States sour cherry yellows has been reported from most states where sour cherries are grown as a commercial crop. It occurs also in British Columbia and Ontario, Canada.

Hosts. Yellows with typical symptom expression occurs in the nursery and orchard mainly on sour cherries of the varieties Montmorency, Early Richmond, and English Morello. By inoculation it has been proven that the virus may be transmitted to a number of varieties of sweet cherry (*Prunus avium*) to blackcherries, pincherries, and chokecherries and domestica, American, and Myrobalan plums. No yellows symptom expression occurs on these, but the virus can be transmitted back to sour cherry with the development of yellows symptoms. It has also been transmitted to several varieties of peach in which symptom expression consisting of necrosis and rosetting appears, rather than the typical cherry yellows symptoms.

Symptoms. The symptoms on Montmorency sour cherry are the appearance of yellow and green mottling of some of the leaves followed by leaf casting. The green color is retained longest along the midrib and larger veins. During the season yellowing occurs in waves, usually the first wave 3 or 4 weeks after petal fall being the most striking. Trees diseased for several years show a much reduced spur system. The trees show a willowy type of twig growth with long bare spaces on the twigs with few fruit-bearing spurs, thus reducing the yield. The fruits produced, however, are larger than those on healthy trees and are of good quality, thus differing from the "little cherry" virus diseases. Symptoms on Early Richmond are much the same as on Montmorency.

Transmission and Incubation Period. Sour cherry yellows virus can be transmitted by budding or other methods of tissue union. Probably insect transmission takes place in the orchard, but the vector is not known. It has been demonstrated that seeds from diseased Mahaleb cherries carry the virus and can transmit it to the seedlings.

Incubation in the field is from 1 to 2 years. It has been possible to observe this period in the greenhouse so that leaf symptoms may appear within 6 weeks after inoculation.

Recently successful transmission of cherry yellows virus to cucurbit hosts has demonstrated that these herbaceous plants may prove of importance in solving the problem of the entities involved in some of the complex virus diseases such as cherry yellows, necrotic ring spot, and tatter leaf.

Control. Experiment stations throughout the sour cherry–growing region have cooperated in establishing yellows-free mother nurseries from which cion wood of the principal sour cherry varieties can be obtained by nurserymen for further propagation. This program includes the propagation of yellows-free rootstock by securing seed from indexed trees of suitable varieties for understock.

New orchards should be planted with yellows-free nursery stock and at a distance from any old orchard in which yellows occur. In bearing orchards where yellows is present in a high percentage of the trees, roguing and replanting is not advised, but entire blocks should be removed when they become unprofitable. In young orchards having few cases of yellows, roguing is advisable.

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Leaf Mottle Virus Diseases of Sweet Cherries

Five virus diseases of sweet cherries have been described in which the main characteristic is some type of leaf mottle. These have been reported only from western United States and from British Columbia, in Canada. The common names given these are (1) mottle leaf, (2) rusty mottle, (3) mild rusty mottle, (4) necrotic rusty mottle, and (5) Lambert mottle. The virus origin of these diseases has been definitely established by transmission studies, and whether or not they are caused by strains of a common virus, they have been differentiated from one another by rather definite variations in symptoms or host relationship. Only a brief account is given here of these diseases, since they are mostly of local distribution and importance.

This presentation of the leaf mottle diseases of sweet cherries is a summarized account of the descriptions published in the *U.S. Department of Agriculture Handbook* 10 by various authors. Some speculations indulged in by the writer are included, for which the various *Handbook* authors are not responsible.

Mottle Leaf. This disease was first noticed in Oregon by Zeller in 1920, and the virus nature of the disease was established in 1935 by McLarty and Reeves. This disease occurs generally in the sweet cherry-growing regions of Washington, Oregon, and Idaho and has been observed in British Columbia, California, Utah, and Montana. It is of economic importance in the foothills or canyon districts of north central Washington and in the Kootenay area of British Columbia.

Bing and Napoleon (Royal Ann) are the two sweet cherry varieties most severely affected. Sour cherries (*P. cerasus*) are susceptible, but symptomless, and peaches seem also to be symptomless carriers of the virus.

The *symptoms* of mottle leaf vary with the variety affected. Severe mottle leaf on the most susceptible varieties consists of irregular chlorotic mottling and a distortion early in the season and increased puckering as the season advances. Leaves may be reduced in size, but there is no defoliation. Fruits may be abnormally small, later than normal in ripening, but not misshapen. Tree growth is stunted with a rosetted effect caused by shortening of internodes between spurs.

The natural vector of the virus is not known. *Transmission* is readily obtained through tissue union. There is evidence that the virus spreads from the wild bitter cherry (*Prunus emarginata*) to nearby cherry orchards. Both severe and mild mottle leaf viruses were transmitted from cherry to peach and back to cherry by Zeller and Milbrath. Symptoms appear relatively early after inoculation, 14 days in the greenhouse and 37 days in the field when inoculated in the spring. Midsummer inoculations do not show symptoms until the following spring.

Rusty Mottle (Severe). This virus disease appears to be confined to sweet cherry orchards in a few counties in Washington. In orchards where found, it is of considerable economic importance. In some orchards 25 to 30 per cent were affected, but the average in all orchards of the region was less than 3 per cent.

All varieties of sweet cherry commonly grown in Washington are susceptible. Peach and sour cherries have been successfully inoculated by budding, but have not been found naturally infected.

The *symptoms* as described by Reeves appear as a chlorotic mottling of the older leaves 4 to 5 weeks after bloom. All the foliage soon becomes more or less affected and develops late-season colors (bright yellow to red) with islands of green. Leaf casting of the brightly colored leaves occurs mainly during the 2 to 3 weeks prior to harvest. Mottling of the remaining leaves becomes more pronounced, and the leaves take on a rusty appearance from which the name of the disease was derived. The *fruit* on diseased trees is smaller, retarded in maturity, and insipid in flavor. When inoculated on peach (freestone) the main symptoms consisted of the development of yellow-orange–colored leaves and the production of various ring spot patterns.

Only artificial *transmission* by tissue grafting has been reported.

The only variations of this disease from leaf mottle seem to be (1) leaf casting, (2) symptom development on the peach, and (3) possibly the intensity of coloration.

Mild Rusty Mottle. This disease ranks first among the mottle-type virus diseases of the sweet cherry in Oregon. In some old orchards it was estimated that more than 50 per cent of the trees were affected. Mild rusty mottle causes some economic loss from the gradual decline in production of the affected trees, although such trees continue to bear fruit for a number of years.

Sweet cherries in general are susceptible, with little difference among varieties. The virus has been obtained from Montmorency sour cherry, and other varieties of stone fruits have proved by inoculation to be susceptible, but most are symptomless. On sweet cherry the *symptoms* consist of a general yellowish-green color which can be noticed at a distance. The foliage takes on a rusty or bronzed appearance by late June or early July. Decline of the tree is indicated by poorer terminal growth and poorer fruit set on spurs. Decline is slower than is the case with trees affected by severe rusty mottle.

The rusty appearance of the leaves is first apparent on the upper surface, but gradually the lower surface shows the rustiness. Some shedding of leaves occurs, but is not pronounced. Fruit of affected trees is somewhat retarded in ripening, and fruits of the Napoleon variety are inclined to be clear yellow and without the red cheeks of healthy fruit.

Mild rusty mottle differs from severe rusty mottle in that decline is slower, the fruit is almost normal in size and quality, and leaf casting is much less. Other differences have been noted, such as the difference in reaction when inoculated on peach, western chokecherry, and oriental flowering cherry. Nevertheless, these two rusty mottle diseases seem closely related.

Necrotic Rusty Mottle. The disease has been found only in Utah where it is of considerable economic importance. A survey in Utah in 1944 showed 20 per cent of 129 orchard blocks infected with from 10 per cent to 46 per cent of the trees showing the disease. Bing, Lambert, and Napoleon are the varieties of sweet cherries affected by necrotic rusty mottle. Black Tartarian is a symptomless carrier.

Symptoms consist of a delayed development of leaves and blossoms in the spring. Leaves which appear healthy until 3 to 5 weeks after bloom develop brown necrotic spots of varying size, shape, and distribution. Defoliation follows on some severely affected leaves. A second defoliation reaches peak expression 2 to 3 weeks before harvest, when from 25 to 85 per cent of the remaining leaves may fall. After harvest the necrotic spots in the remaining leaves frequently fall out, producing a conspicuous shot-hole effect. Senescence of leaves on diseased trees occurs 2 to 4 weeks earlier than on healthy trees. Some of the buds on the leaf spurs are killed, resulting in bare branches with terminal tufts of foliage. In advanced stages the older branches are killed and numerous water sprouts may develop from the trunk or lower portions of the main branches.

Transmission of the virus is easily obtained by tissue union on Mazzard seedlings and Bing, Lambert, and Napoleon varieties of sweet cherry. No insect vector is known, but there is some evidence of natural spread in the orchard. Diseased nursery stock is probably the most common means of distribution.

Lambert Mottle. This virus disease is described by Lott as occurring only in the Okanagan Valley of British Columbia in Canada. It is of minor economic importance because of its local occurrence and the fact that only a low percentage of the trees is affected.

As the name indicates, the disease is found mainly on Lambert. There are little or no symptoms on Bing and Napoleon, although these varieties are known to be susceptible.

Symptoms on trees on which the disease is well established consist of failure of the buds on terminal shoots to develop. The development of the other leaf and flower buds is both late and irregular. In June a yellowish interveinal mottle begins to appear on the older leaves. This is followed by numerous small spots of a purplish color, which later becomes brown. About half of the affected leaves fall prematurely, but this may vary from year to year. Diseased trees set only a light crop, and sometimes many of the fruits do not reach maturity. The disease becomes progressively more serious for several years with death of twigs and larger branches.

The virus is readily transmitted by tissue union, but no vector is known. It spreads slowly in established orchards.

There is some indication that Lambert mottle may be a strain of the disease described as necrotic rusty mottle in Utah. Recently a more virulent and quick-acting strain of the Lambert mottle has been observed in the Okanagan Valley which has symptoms much like those of necrotic rusty mottle.

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Prune Dwarf

This virus disease, primarily on prune and plum, has been called prune mosaic, Fellenberg mosaic, willows, and shoestring of Italian prune. It is known only from New York, Ontario, and British Columbia. It is of minor economic importance at present, but is of potential importance since strains of the virus occur on sweet and sour cherries.

The *host* range of the virus by natural transmission or inoculation is extensive, but serious injurious effects are confined mainly to the Italian prune and to a lesser extent to Lombard plum. Symptoms are produced on peach and on Mahaleb cherry when inoculated. In damson plum, domestica plum, Japanese plum, Myrobalan plum, sweet and sour cherries, and apricot, the virus occurs without symptoms. The disease has been found in nature on Bradshaw and damson plums, Italian prune, and sweet and sour cherries.

Symptoms. On *plum* (Italian prune) typical symptoms consist of small, narrow, straplike, rugose, distorted leaves. Terminal growth is reduced so that the internodes are shortened. The normal blooming is followed by abortion of pistils, and few fruits mature. However, the few fruits which mature are larger than normal and of good quality. The symptoms on Lombard are much the same as those on Italian prune but less severe. It has been demonstrated that there are several strains of this virus and that the symptom expression depends upon the strain under consideration.

When *peach* is inoculated the resulting symptoms are (1) a slight delay in foliation and (2) wavy leaf margins and a tendency for the leaves to roll upward and to be stiff and erect. A loose rosette effect due to shortening of internodes is noticeable with some strains.

On *sweet cherry* the symptoms consist of ring spotting and oak leaf patterns. The symptoms are masked after the first season of infection. Some reduction in yield has been noted, but with no marked effect on fruit size or quality. On *sour cherries* (Montmorency) some of the early leaves may show ring spotting, necrosis, and flecking. The symptoms may disappear later in the season, and in subsequent years there is no evidence of the disease.

The symptoms on peach resemble those produced by rosette mosaic, but there appear to be sufficient differences to warrant separation.

Transmission. The insect vector is not known. The virus can be transmitted by grafting and budding.

Control. Where Italian prune is grown in an orchard, careful annual inspection of the trees is recommended, followed by prompt roguing of diseased trees.

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Other Virus Diseases of Stone Fruits

In the preceding pages the most important stone fruit virus diseases have been treated in some detail. About forty additional diseases proved by transmission experiments to be of virus origin have been described. Many of these have been inadequately studied and may be strains or forms of previously described virus diseases or may be symptom expression of a virus complex. Most are local in their distribution or not especially destructive.

A brief statement concerning the host range, geographical distribution, symptoms, and special characteristics of a few of these diseases is all that is warranted in this text.

Red Suture. Red suture occurs on peach and the Abundance variety of Japanese plum. It is known to occur only in Michigan and Maryland, but seems to be of importance only in certain areas in Michigan and probably was introduced in Maryland on nursery stock. In the region where it is established it causes an annual loss of about 3 per cent of the trees. The fruit ripens several days prematurely with softening, swelling, and red splotching on the suture, while the remainder of the fruit is hard and green. On the suture side the flesh is coarse and exceptionally watery. Trees with red suture are off color and take on autumnal coloration prematurely. Two-year-old branches send out short spurlike shoots with a resulting crowding and clustering of the leaves along the branch. Diseased trees continue to live over a normal period of years, but the fruit is worthless.

When transmitted to Abundance plums the symptoms are identical with those of peach yellows.

Transmission has been accomplished by tissue grafting only. No insect vectors have been discovered.

In most areas red suture can be controlled by periodic inspection and eradication. Growers should be certain that trees purchased were propagated from budwood known to be free of disease symptoms.

Rosette Mosaic. Known only from a limited area in Berrien County, Michigan and a few scattered cases in New York, this disease is of little economic importance. It is confined to peach and plum. The symptoms of rosette mosaic are similar in some respects to those of prune dwarf, sour cherry yellows, and peach mosaic, but differ from all these in several respects.

On peach, the symptoms are delayed foliation, chlorotic mottling of early-formed leaves generally accompanied by deformation, and a shortening of internodes, giving a rosette appearance to the shoots. Affected trees have a darker green color than normal. There are no distinct symptoms on the fruit, but production is reduced.

An especially interesting feature of this virus is that it is apparently soil-borne. Healthy trees planted in soil from around diseased trees or planted in locations previously occupied by diseased trees often become diseased. It is possible that unknown vectors in the soil may be responsible for apparent soil transmission. It is readily transmitted by grafting.

Control consists of eradication and avoiding replanting in soil from which diseased trees have been removed.

Yellow Bud Mosaic. This disease of peaches and apricot is known only from Solano and Yolo Counties in California. Trees of Elberta, Lovell, and Muir peach are reduced to an unprofitable state in a few years. Natural infection has been found on almond, apricot, and peach, while a number of varieties of peach have been artificially inoculated, and the virus has been transmitted to almond, apricot, rose, Japanese kerria (*Kerria japonica*), and Myrobalan plum.

On peach symptom expression consists of pale yellow, oblong, feather-edged blotches along the midvein of the leaves, with distortion of the leaf blade. In advanced stages of the disease defoliation occurs, and the basal portion of the affected shoots may be bare. The remaining leaves are clustered at the tips of the branches, giving a rosette effect.

The virus can be transmitted by tissue grafting. In orchards, spread occurs from infected trees to adjacent trees only, but no insect vector is known.

Muir Peach Dwarf. Of no great economic importance, this virus disease is of interest because of several peculiarities. It is known to occur naturally on only one variety of peach, the Muir, and only from California, where this variety is grown as a superior drying peach. It is known to have been present in certain orchards as early as 1920, but it was not until 1936 that transmission experiments were initiated to prove this disease of virus origin. The symptoms of the disease suggested that the trouble might be either peach rosette or phony, but a closer study indicated that neither of these viruses was involved.

When in full leaf the dwarfed trees show a profusion of large, flattish, dark green leaves, rather closely appressed on short twigs, while on healthy trees of this variety the leaves are lighter green and curved upward along the midrib and spaced evenly along the long shoots. During the dormant season a whorl of short, stubby lateral branches on the terminal growth give a loose witches'-broom effect. Fruit production falls off with increasing age of the disease, and the fruit in advanced stages is larger than normal and misshapen.

The Muir variety seems to be the only peach naturally infected. Elberta peach trees grown for a number of years in close proximity failed to show the presence of the virus when indexed on Muir trees. That the virus can be transmitted by grafting to other varieties of peach and to almond, apricot, cherry, and plum was determined by budding back to Muir seedlings from these inoculated varieties. Typical symptoms developed on the Muir seedlings, although no symptoms appeared on some of the varieties originally inoculated. Some peach varieties such as Paloro Alexander and Lovell were severely injured by the virus when graft-transmitted, but Elberta and most other stone fruits were symptomless carriers.

Control is relatively easy, since diseased Muir trees are easily detected and can be eradicated in an early stage of the disease. Since there seems to be no natural spread to other varieties, there is no danger of symptomless carriers. Budwood of the Muir variety, of course, should be obtained from a region known to be free of the disease.

Wart. This peculiar virus disease was first reported from Idaho in 1939. It is now known from several areas in Idaho, Oregon, Washington, and New Mexico, and in San Bernardino and Riverside Counties in southern California. Generally, the number of diseased trees is small, and on the whole the disease is of minor importance. Diseased trees show a high percentage of warty fruits and are worthless. The virus has no effect on the development of the tree.

The symptoms appear on the young peach shortly after shuck fall as bleached bumps near the tip end and may involve half or more of the fruit. When severely diseased, the fruit is dwarfed and misshapen, but slightly infected fruits are normal in size. Cracking and russeting with conspicuous gum exudation often result as the fruit enlarges. The warty tissue is rather superficial, but the underlying flesh is coarse and filled with gum pockets.

Warty fruit caused by other agents such as insects and the peach leaf curl fungus may cause confusion in attempting diagnosis of this condition.

The virus has been transmitted by grafting to some varieties of sweet cherry where mottled leaf symptoms occur. Warty fruit tissue grafted into young peach tree trunks resulted in the development of wart in these trees.

Prompt removal of diseased trees and use of budwood from wart-free trees are recommended as control measures.

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Yellow Leaf Roll of Peach. A new virus disease of peach was discovered in 1950 in the peach bowl area of north central California. Three years of inspection during which over 7 million trees were examined in 22 counties in California has indicated that yellow leaf roll is confined to Butte, Placer, Sutter, and Yuba Counties and is not known to occur outside California. A total of about 3,000 trees were removed during the three-year period.

Leaf symptoms consist of a marked yellowing and inward rolling of the leaf edges with reddish to brown coloration. Defoliation occurs before normal leaf drop. The disease may start on one or more limbs and advance upward, with some limbs showing normal foliage. Diseased limbs usually die following the first year of infection.

In some respects this disease resembles the X-disease of peach and may prove to be a strain of this virus. It has been successfully transmitted by tissue union. The geminate leaf hopper (*Colladonus geminatus*) is capable of transmitting this, as well as the western X-disease virus. Its host range is not known, but it seems to be limited to the peach in the area where present. It has been found on cling varieties only, but has been artificially transmitted to freestone varieties.

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Plum Pox

In southeastern Europe plums are the most important fruit crop. It is estimated that plums constitute 80 per cent of the fruit-growing industry in Yugoslavia. By far the most important virus disease of this crop is "plum pox." Todorovic, in 1952, reported that 50 per cent of the trees in Serbia were infected and that the disease had caused the complete arrest of plum cultivation in some areas. Christoff states that the disease occurs in all plum-growing areas in Bulgaria, and severe losses are sustained.

The geographical distribution of plum pox is uncertain since it has been confused with a number of other virus diseases of stone fruits. Because of this confusion the treatment which follows will be based on the investigations of the plant pathologists of Yugoslavia and Bulgaria.

Symptoms. According to Atanasoff the infected tree appears little different from the healthy tree as to vigor of growth. The *leaves* show various types of mottling, depending on the variety and season. In general the mottling is indistinct and consists of light green to yellowish-green blotches which first appear after the leaves are fully expanded. Christoff, in a description of the disease in 1938, referred to it as the "broad-streak and ring spot variegation—plum pox" and described broad bands or stripes on the leaf blade and on some varieties a ringlike effect.

The most pronounced symptoms occur on the *fruit.* Christoff in 1947 states that on infected plum trees the disease causes a fruit drop 30 to 40 days before ripening, and very little fruit remains on the tree. Most of the fruit is poxed with discolored, gummy pulp underneath. The diseased fruit remaining on the tree ripens 10 to 15 days before the normal ripening period.

Hosts. According to Christoff the "true" plum pox virus attacks only plum (*Prunus domestica* and *P. institia*), apricot (*P. armeniaca*), and wild plum (*P. cerasifera*), and *P. triloba.* "All efforts to transmit the disease from plum, apricot and wild plum to hundreds of cherries, (*Prunus avium*), sour cherries (*Prunus cerasus*), mahaleb (*Prunus mahaleb*), peach (*Prunus persicae*) and Almond (*Prunus amygdalus*) nursery stock and over one hundred varieties of them so far have given no results" (Christoff, 1947). Thus the various reports of this virus on hosts other than plum and apricot are probably based on questionable diagnosis.

Transmission. The virus is transmitted easily by budding and grafting, and this probably accounts for its wide distribution in Yugoslavia and Bulgaria where proper care has not been taken by nurserymen to obtain budwood from virus-free trees. The virus is not seed- or soil-transmitted. An aphid, *Anuraphis helicrissii,* is a vector and accounts for the natural spread of the disease. The incubation period in the host is from 9 to 13 months.

Control. Eradication of diseased trees is the only known method of control. The only hope of control in southeastern Europe appears to be rigid nursery inspection and regulation and the development of plantings of virus-free trees from which nurserymen can obtain budwood for future tree production. Only disease-free understock should be used. It has been found that most French seedlings are free of the virus.

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Stone Fruit Virus Diseases of Minor Importance

Rasp Leaf of Sweet Cherries. First observed in Colorado in 1935, rasp leaf was proved of virus origin in 1942. Later it was found in British

Columbia, Canada, Idaho, Washington, Montana, Utah, and California. The disease is of minor importance in most regions, although in Colorado there is evidence of rapid spread.

The most characteristic *symptom* is the outgrowths on the lower surfaces of the leaves (enations). These may be leaflike or toothed in character. The growths originate between the veins and radiate from the midrib toward the leaf margin. Severely affected leaves are small, narrow, and distorted. Damage to diseased trees consists of retarded growth and reduction in the size of the crop.

Transmission has been effected only through graft and bud inoculations. The incubation period may range from 9 months to 2 years.

Cherry Rugose Mosaic. Known only from California, rugose mosaic is of minor importance and does not appear to spread rapidly in orchards where it occurs. Natural infections have been recognized in Black Tartarian, Burbank, Napoleon, Black Republican, and Rockport varieties of *Prunus avium*. The virus was transmitted artificially to almond, peach, and Mahaleb cherry, with mild symptoms resulting.

The most obvious *symptom* consists primarily of a general chlorosis of the leaf blade midway between the midvein and leaf margin. Affected leaves usually shrivel and drop by late June or July. The yield of fruit seems to be somewhat reduced.

The virus is transmitted by grafting.

Other stone fruit diseases of minor importance are as follows: On *peach*, "Asteroid spot," "calico," and "low twig," are mostly locally distributed and of no economic importance. On *plums* and *prune*, "diamond canker" is of limited distribution in California, and "line pattern" is reported from the north central states, Washington, and Oregon in the United States, and from Ontario, Canada. On *apricot*, "ring pox," first observed in Colorado, is also known from Washington. [*U.S. Dept. Agr. Agr. Handbook*, **10** (1951).]

Viruslike Diseases of Stone Fruits

Several diseases of stone fruits have symptoms suggestive of the transmissible virus diseases, but to date efforts to transmit these diseases have failed. These disorders, in some cases, may be of genetic origin.

Sweet Cherry Crinkle Leaf. This disease was described in 1930 under the name "unproductive cherry" [C. F. Kinman, A study of some unproductive cherry trees in California, *J. Agr. Research*, **41**:327–335 (1930).] Subsequent reports showed the trouble to be so common in Black Tartarian sweet cherry that its known occurrence coincides with this cherry variety in the major sweet cherry sections of western United States and Canada.

Crinkle leaf is of considerable *economic importance* since affected nursery trees planted in commercial orchards are usually not recognized until the healthy trees begin bearing. Since the diseased trees are worthless, the expense of maintaining the trees to bearing age is a decided loss in addition to the necessity of replanting. Surveys in Washington show that from 4 to 6 per cent of the sweet cherry trees in that state are affected. Bing and Black Tartarian are the chief commercial varieties subject to this malady. A number of other sweet cherry varieties have been listed as showing definite evidence of crinkle leaf.

Symptoms show as variously misshapen and mottled leaves. Margins of the leaf are often indented and abnormally serrated through failure of the blade tissue to develop. Affected trees bloom in a normal manner, but many of the blossoms are smaller than normal and are visibly defective. Few pistils develop fruit, and such fruits are small, pointed, and often with a raised suture. It is common for trees to have both normal-appearing branches and those affected with crinkle leaf.

Efforts to transmit the disease to healthy trees were unsuccessful, although the inserted grafts continue to show the disease. Diseased trees topworked with normal cions also failed to transmit the disease to the healthy cion wood.

Deep Suture. This viruslike disease has been reported as fairly common on the Bing variety in California, Idaho, Montana, Oregon, Utah, and Washington in western United States and in British Columbia, Canada. The economic loss is from the refusal of packers to accept the affected fruit. Since many trees show malformed fruit on only a few branches, the loss may not be great, especially since in some seasons even fruit on affected limbs may not be seriously malformed.

Bing trees affected with deep suture have variable percentages of malformed leaves and fruit. *Leaves* are often narrow and elongated with rough, but not puckered, surfaces. The leaf margins may have fewer serrations than normal. Pronounced internal chlorosis such as occurs on trees affected with crinkle leaf does not occur, although there may be faint light green areas during late spring which become less evident later in the season.

Affected *fruits* have a pronounced depression on the suture side, are normally rounded at the end, and show little, if any, decrease in size.

Attempts to transmit deep suture by a number of investigators have failed. Various methods of transmission have been tried without success.

NONPARASITIC DISEASES OF STONE FRUITS

Deficiency and Excess Nutrient Troubles of Stone Fruits

Nonparasitic disorders of stone fruits are mainly caused by deficiency or excess of one or more of the essential mineral nutrients in the soil. The symptoms are such that the diseases are often confused with those caused by viruses or even parasitic organisms. Leaf spotting, chlorosis, and tip or marginal burn are often ascribed to climatic factors or root diseases, but are frequently symptoms of mineral deficiency. A brief description of some of these troubles is here given as a guide for diagnosing such disorders found in the field.

The material here presented is taken mainly from the *U.S. Department of Agriculture Handbook* 10 (1951), where various authors have treated these and other mineral-deficiency troubles. No attempt is made to include many other disorders which may appear as a result of deficient or excess elements in the soil.

Nitrogen Deficiency. Lack of available nitrogen is the most common and important cause of deficiency disorders in stone fruit. The peach especially is a nitrogen-demanding tree, and ill effects result when this element is lacking. *Symptoms* consist of yellowish-green leaves, beginning on old, mature leaves and progressing toward the tip. Purple or red spots often appear on the blade of the leaves. These spots in severe cases become brown and necrotic in midseason. Defoliation of basal leaves may occur. Twig growth is checked, and the twigs remain slender. Leaf fall is usually several weeks in advance of trees supplied with sufficient nitrogen. The fruit is usually much reduced in size unless excessively thinned.

Phosphorus Deficiency. The older leaves are mottled with light green areas between dark green veins. Progressive defoliation of mottled leaves from the bases toward the tips of the twigs occurs as the season advances. Purplish pigment develops in the stems and leaf petioles during cool summer weather. The mottled leaves are likely to be confused with some of the virus mottle diseases. The symptoms are more likely to occur on peach than on any of the other stone fruits.

Potassium Deficiency. The most striking symptoms on the *peach* are the dead areas on the leaves, varying in size from small spots to large irregular areas and often marginal injury. Twigs are likely to be slender, and fruit buds sparse. The foliage in the spring is usually a lighter green than on trees having sufficient potassium.

On *cherry* the lighter green character is noticeable and the leaves have a tendency to curl upward. On *plums* the symptoms develop rather late in the season. In July or August the leaves begin to lose their normal color, usually fading first at the margins. As chlorosis progresses, the older leaves roll inward toward the upper surface. These symptoms appear mainly on the peripheral portion of the tree. The fruit may be delayed in ripening, and on young trees there is a marked dwarfing.

Magnesium Deficiency. On peach, the symptoms consist of light gray or fawn-colored spots in the area between the veins. In severe cases the spots may enlarge and the affected area may include the margins of the leaves. The leaves of severely affected trees are shed from the base toward the tip of the twigs, and only a tuft of the tip leaves may remain.

Manganese Deficiency. Noted on most of the stone fruits, this deficiency is manifested by the symptoms on the leaves. The midrib and main veins with adjacent bands of tissue of varying widths remain green, while the interveinal and peripheral areas are chlorotic. In Illinois there is some evidence that internal bark necrosis of the peach may be caused by *excess* available manganese. [H. H. Thornberry, Manganese toxicity in peaches. (Abstr.), *Phytopathology,* **40**:29 (1950).] This condition may result ultimately in the formation of large dead areas on the trunk and branches of the tree.

Zinc Deficiency. Experiments on the control of bacterial spot of peach called attention to the fact that when a zinc sulfate–lime spray was applied, the foliage took on a darker green color than that of the unsprayed trees or those receiving lead arsenate–lime applications. Sweet cherries are the most susceptible of the stone fruits to zinc deficiency. Plums, peaches, apricots, and almonds have been observed to be injured when zinc is not available. Zinc deficiency has been a serious problem in many of the western states on stone fruits.

Symptoms consist of irregular splashes of yellow occurring between the veins, especially on leaves of the new growth. In general, the foliage is of a lighter green than on unaffected trees. Leaves at the tips of new growth are small, more or less crinkled, and chlorotic. There is a progressive defoliation from the base to the tip of the twig. In some cases, the internodes are shortened, so that a rosette effect results. Terminal die back occurs in some stone fruits, such as the sweet cherry.

Iron Chlorosis. Iron deficiency is common in most stone fruits as well as other tree fruits. The most evident symptom is the general paleness of the foliage resulting from the lack of chlorophyll. Chlorosis appears first at the tips of growing shoots. This may be the only evidence of iron deficiency the first year, but if not corrected, more and more of the leaves will show the condition in succeeding years. On trees affected with iron chlorosis, the leaves are chlorotic when formed. The degree of yellowing varies from complete absence of green to light yellowish-green areas between the veins. There is usually a narrow band of green along the midrib, main veins, and veinlets. The symptoms of iron deficiency are not always specific and may be confused with other causes such as potassium or magnesium deficiency. Since iron deficiency is quickly corrected by spray applications containing available iron, it is possible to determine whether or not the chlorotic condition is due to lack of this element. Sprays should be applied early in the season since the condition can be corrected much more rapidly at this time than later in the year.

Boron Deficiency. Plums, peach, and apricot are stone fruits likely to show boron deficiency. In California *Prunus domestica* varieties President, Giant, and Diamond are affected, while varieties of Japanese plums (*P. salicina*) growing adjacent to affected domestica show no evidence of the disease. In British Columbia peaches and apricots are affected.

The *symptoms* on plums are mainly on the fruit. Brown sunken areas in the flesh may consist of a single spot or may involve most of the fruit. The flesh beneath is brown and firm. [This condition resembles the virus disease plum pox (see page 299).] The fruits color earlier than normal and drop.

In British Columbia on *peach* the outstanding symptoms are failure of buds to break in the spring and the dying of twigs, branches, and even entire trees.

Boron-excess Injury. In sections of California where stone fruits are irrigated by water high in boron, serious injury may occur. On plum, peach, apricot, and almond the principal injury is to the shoots of the current season's growth. The shoots die back at the tip, and the bark cracks and becomes corky. The lateral buds on injured apricot, plum, and peach shoots start to grow, only to be killed back later. This may result in a "knobby" appearance of the apricot twig.

Spray Injury to Stone Fruits

A few of the outstanding types of spray injury to stone fruits are here presented, but no attempt is made to discuss these in detail.

Lime Sulfur Injury. When lime sulfur was first introduced as a summer spray for such diseases as apple scab, experiments were undertaken to apply it to stone fruits. It soon became evident that serious injury to the foliage and fruit of peach and some varieties of plums would result when the concentration used on apples was applied. Later, the safer self-boiled lime sulfur and wettable sulfurs were substituted.

Injury from lime sulfur appears on the leaves and fruit. On the leaves, marginal and tip burn is the most conspicuous effect, but often large bleached areas on the blade between the veins develop, especially following hot weather. On the young fruit, the side of the fruit exposed to the sun is bleached, and a sunken area results with the enlargement of the fruit.

Lead Arsenate Injury. For over a half century lead arsenate was the standard spray for the control of peach insects. While it was realized that some injury might result, the peachgrower had no other effective insecticide to substitute. Later, with improvement in the formulation of lead arsenate, some decrease in injury was obtained. The substitution of dusts for liquid sprays was found to eliminate much of the injury, but dusts were not as effective as the liquid sprays. It was not until the introduction of zinc sulfate–lime as a peach spray that the amount of invisible injury to the peach by lead arsenate was realized. While the zinc sulfate spray was introduced primarily for the control of bacterial spot, its effect as a safener for lead arsenate was found to be its most valuable function.

Lead arsenate injury appears on the foliage, twigs, and fruit. On the leaves, brown or purple spots of varying sizes develop, usually from 10 days to several weeks after the spray is applied. Marginal and tip injury may occur. On the fruit, sunken areas similar to those resulting from lime sulfur sprays sometimes appear, especially on the fruit of trees in poor vigor. The injury on the twigs consists of purple or brownish oval areas around the buds on the current season's growth. This injury may be superficial or, in extreme cases, may cause death of the bark tissue to the cambium. "Invisible" injury is apparent when trees receiving a number of lead arsenate–lime sprays are compared with those sprayed with a noninjurious material. The leaves of the lead arsenate–sprayed trees are a duller green and smaller. One of the most serious aspects of lead arsenate injury is early defoliation. Even though no sprays are applied after 2 or 3 weeks before harvest, leaf fall may be extensive during September and October, while trees sprayed with other materials retain their foliage until the first heavy frost. This early defoliation is not so noticeable on trees dusted with lead arsenate–containing dusts.

Copper Spray Injury. In dry climates Bordeaux mixture has been safely used on several of the stone fruits, including the copper-sensitive peach, but in regions subject to frequent rains most copper-containing sprays are unsafe on peach.

Copper injury usually appears on the foliage of the peach following a rain after the spray is applied. Numerous small red or purple spots appear on the leaf blade. In a day or so the defoliation starts, and often the tree is almost completely defoliated. If the weather continues dry and hot for a period of a week or more after the application, no serious injury may result. A shot-hole effect occurs on some varieties of plums following the appearance of purplish spots on the leaves. Other varieties of plums are not affected at all.

Fluoride Injury. At one time certain fluoride sprays were used as insecticides on peaches. Sodium fluosilicate was experimented with for the control of bacterial spot of peach. While these spray materials have been abandoned as peach sprays, the peculiar injury produced is of some interest. The condition brought about by the fluoride sprays was called "soft suture" and resembles in many respects the virus disease of peach known as "red suture." The fruit matures several days ahead of normal ripening. The suture takes on a yellowish red to deep-red color and softens even while the remainder of the peach is quite firm. No decided difference is noted in the taste of the fruit, but the soft suture is easily bruised in packing, and the fruit becomes unsightly.

Heat Spot, or Kelsey Spot, of Plums

In regions where high temperatures (above 105°F) occur over a period of several hours during the ripening period of plums, a diseased condition develops which received the name "Kelsey spot" from the variety on which it was first observed. It has been reported from both eastern and western North America and from South Africa and Australia.

The spot usually occurs on the distal end of the fruit, but may occur on any part. Slight injury appears as a shallow, reddish depression, underlaid by brown necrotic tissue. In severe cases the affected area is purplish red and the necrotic tissue beneath may extend to the pit. "The spot differs from sunburn in having sharp margins, depressed centers, and a purplish-red color in contrast to the diffuse margins, normal shape and brown color of sunburn."

It was definitely established by Dippenaar in South Africa and by Proebsting in California that the spot is caused by high temperatures. No organisms have been found associated with the disease. The condition can be duplicated in the laboratory by subjecting plums at a certain stage of maturity to high temperatures. Kelsey plums exposed to 107°F for 15 hours gave 10 per cent damage, and for 72 hours, 60 per cent. In the field, shading prevented the occurrence of the spotting, and cover crops in the orchard reduced the temperature below the critical point, while in neighboring blocks having no cover crop, as high as 1 per cent of the fruit showed the spotting.

Some varieties are much more subject to heat spot than others. In California it was observed that widespread injury occurred on Japanese plums, whereas there was little injury on European varieties in the same orchard. The time of ripening in relation to high temperatures is also a factor since the fruit is subject to heat injury only during a relatively short period near the time of maturity.

The use of summer cover crops in the orchards where this trouble occurs appears to be the best method of control.

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STONE FRUIT DISEASES INCITED BY NEMATODES

Root Knot

In regions where short, mild winters prevail, root knot nematodes on stone fruits may cause severe damage. In North America the northern peach-growing areas are rarely affected. The principal damage is to peach trees in light, sandy soils. Root knot is found also on plums, cherries, and almonds, but apricots rarely show the disease.

Symptoms. Trees may show no definite symptoms aboveground when the nematode attack is light, and in some cases, mildly affected trees may recover after the initial weakening. Heavily infected peach trees are stunted, with foliage lighter in color than normal. The tree may finally die, but this is thought to be due to other factors, following the general weakening of the tree by the nematode attack. The main symptom, of course, is the presence on the roots and rootlets of the typical galls. These galls, however, may be confused with swellings caused by insects, or with crown gall. The root knot galls may be spindle-shaped enlargements or irregular globular growths of variable size, in peach sometimes being an inch across. In some cases, a number of rootlets may arise at or near the enlargements.

Geographic Distribution. Root knot on stone fruits is world-wide in its distribution, but it becomes severe mainly in warmer climates with mild winters.

Economic Importance. Losses result from the general weakening of the trees, which then may succumb to drought or other adverse conditions. Weakened trees also are much less productive. The distribution of nematode-infected trees by nurseries or planting in heavily infested soil may entail a serious loss since the orchardist is saddled with the expense of the planting and care of the trees before he discovers the disease. Furthermore, since the nematodes may survive on other crops, the introduction of the nematodes may result in a persistent problem.

Causal Organism. As has been pointed out in the general discussion of nematodes in Chap. 1, the root knot nematodes were all regarded as a single species until 1949 and were known as *Heterodera radiciola,* or later as *H. marioni.* Chitwood then demonstrated that actually there were several morphologically distinguishable species for which he reestablished the older generic name *Meloidogyne.* Two of these species are able to reproduce in roots of peach: *Meloidogyne incognita* (Kofoid and White) Chitwood and *Meloidogyne javanica* (Treub) Chitwood. According to Chitwood, Specht, and Havis, *M. incognita* is the more widespread on peaches in this country; on various crops it is generally distributed throughout the southern United States and Mexico and it overwinters as far north as New Jersey. These workers also report that while *M. hapla* Chitwood and *M. arenaria* (Neal) Chitwood do not reproduce in peach roots, they are capable of inflicting serious injury to young trees when planted in soil heavily infested with them.

Hosts. The various species of root knot nematodes have been reported on over 1,800 species of herbaceous and woody plants belonging to numerous families.

Control. If it were possible to plant only nematode-free nursery stock in noninfested soil, control might be relatively simple. In any case, it is wise to obtain trees from nurseries in more northern regions or from those having a reliable inspection service to reduce the likelihood of planting trees that are already infected. Actually, however, many of the older stone fruit–producing sections of the country now have root knot nematodes so widespread in the soil that planting healthy trees gives no assurance of freedom from trouble. Temporary nematode control that can come from treating the soil with one of the nematocidal fumigants before making a new planting may give the trees a good start. Such soil disinfestation is treated elsewhere (page 23).

Use of root knot–resistant understocks affords a useful method of control. Commercial varieties of stone fruits are propagated mostly by budding. This makes it feasible to use nematode-resistant stocks when such are found and are proved to be compatible with the fruit variety to be grown. The most promising understock for peaches at present is variety S37. This is highly resistant to both *M. incognita* and *M. javanica.* For a time it was thought that Yunnan and Shalil were suitably resistant, but then it was learned that in some orchards they gradually became heavily galled. After the separate species of *Meloidogyne* had been recognized, it became apparent that these varieties were resistant to *M. incognita* but not to *M. javanica* and that where the latter species occurred these understocks were not suitable. It has been suggested that highly resistant understocks better adapted than S37 to some orchard conditions may possibly be developed by breeding.

Even when resistant understocks are used and the soil is fumigated before planting, it is important to use only resistant cover crops in the peach orchard. Susceptible cover crops may allow various species of root knot nematodes to multiply until they damage even resistant roots in which they are unable to reproduce. McBeth and Taylor, in 1944, reported an experiment in southern Georgia in which peaches were grown with susceptible cover crops (Whippoorwill cowpeas in summer and Austrian winter peas in winter) in comparison with highly resistant cover crops (*Crotalaria spectabilis* in summer and oats in winter). The resistant cover crops gave less galling of roots, greater growth of peach trees, and vastly greater yields of fruit. The 3-year average fruit yield per tree was 7.7 lb for the susceptible cover crop and 44.5 lb for the resistant; in the fourth year the comparison was 9.8 lb versus 142.2 lb per tree. In the same experiment, bare fallow and the use of a "trap crop" gave intermediate results, but neither can be recommended as practicable. This experiment was with trees on susceptible stocks, but a few trees on Yunnan stock also showed improvement with the use of resistant cover crops.

Root Lesion, or Meadow, Nematode Injury

Meadow, or root lesion, nematodes belonging to the genus *Pratylenchus* attack the roots of a large number of herbaceous and woody plants. The effect of the attack of these nematodes on stone fruits has been given considerable attention, especially in California. Some question has arisen as to the exact role of the nematodes in the decline of stone fruit trees. Is this decline primarily the result of the feeding of these nematodes or do they serve to open the way for invasion of other harmful agents such as wound-invading fungi and bacteria? While this question has not been satisfactorily answered, it has been proved that where the nematodes are found in great abundance on the roots of fruit and nut trees, the general effect is evident in the stunting of growth, die back, and reduced production. Peach trees planted in uninfested or disinfested soil show an increased growth and absence of die back, but this may be due in part to other factors.

Symptoms. No symptoms on the aboveground part of the tree serve to distinguish root lesion nematode injury from that of other agents. In sweet cherry, Serr and Day state that affected trees "make weak, sparse growth followed by dying back of twigs or branches at the end of the main limbs." On such trees many of the small fibrous roots are killed.

Root symptoms begin with the development of minute brownish lesions in the primary cortex of young rootlets. The youngest lesions usually follow the outlines of a very few dead cells. Such lesions develop rapidly where the nematodes enter the root. During entry and after they are within the cortex, these nematodes kill cells as they move about to feed and reproduce. After a time, one lesion may contain adults, eggs, and larvae of all ages. The lesions gradually enlarge and may coalesce to cover much of the rootlet. Bacteria and fungi from the soil also enter such lesions, and it becomes difficult to determine just how much of the final injury is attributable to the nematodes alone.

Gradually, if there is a heavy infection, rootlets are killed. An abnormal number of branch roots may push out and in turn be killed, resulting in miniature witches'-brooms, or brushes of brown rootlets. Usually the nematodes invade only the primary cortex, but when sometimes they invade the rays they may cause narrow sunken lesions in roots that have undergone considerable secondary thickening.

Causal Organisms and Hosts. Early reports listed *Pratylenchus pratensis* or *P. musicola* as the causal agent. It now appears that *P. vulnus*

Allen and Jensen is the predominating species on trees in California. In Ontario, *P. penetrans* (Cobb) occurs on peach. Both species have very wide host ranges. For a further description of the root lesion, or meadow, nematode, see Chap. 1.

On stone fruits, *P. vulnus* has been reported on sweet cherry, Mahaleb and Mazzard cherries, peach, plums (including the Myrobalan and Marianna), and almonds.

Control. Bokhara and Yunnan 55885 peach rootstocks appear to be resistant, according to Day and Serr. Mahaleb rootstocks for cherries show evidence of resistance or tolerance, as do apricots.

For control by soil treatment see Chap. I and also Nematode Control of strawberry.

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CHAPTER 8

DISEASES OF BRAMBLES

Raspberries, blackberries, and dewberries (trailing blackberries) are generally grown throughout the Temperate and subtropical zones for commercial production of fruit. In addition, these crops are often included in home gardens, and in many areas a substantial harvest is obtained from the wild plants growing in woods and wastelands.

Botanically, brambles are members of the family Rosaceae and the genus *Rubus*. They are shrublike or trailing perennials, usually with prickly stems. The term "cane" is used to indicate the individual stalk. The "fruiting cane" is developed during the season previous to fruiting and dies some time after fruiting. New canes arise either from "suckers" from the roots (blackberry and red raspberry) or from tips of the shoots which develop roots (black raspberries and trailing blackberries). The edible "berry" consists of a cluster of drupelets borne on a receptacle, which in the raspberry remains attached to the pedicel when harvested, while in the blackberry it is detached with the fruit.

A red raspberry (*Rubus idaeus strigosus*) similar to the European red (*R. idaeus*) is native to America. The common cultivated varieties of the red-fruited types originated from these two by selection and breeding. The black-fruited or blackcap raspberry (*R. occidentalis*) is native to North America, and the varieties now grown are derived from this species. Crosses between these species have yielded new types such as the purple-fruited varieties.

Over 100 species of blackberries have been described, but most of the cultivated varieties originated from the common upright native blackberries of America, or from the trailing species, or by hybridization of the upright and trailing. Some hybrids are crosses between blackberries and raspberries.

Blackberries and raspberries have most of the diseases in common, but there may be considerable difference between the two as to the amount of damage sustained. For example, most of the numerous virus diseases of raspberries are either not known on the blackberries or are masked. In the treatment which follows, the host on which the disease is of greatest importance will be selected. Because of the great confusion as to the correct Latin names, usually only the common names "red raspberry," "black raspberry," and "blackberry" will be used.

Raspberry Anthracnose

"Bramble anthracnose" would be a more appropriate name for this disease since it is common on all bramble species and varieties. Other common names are "cane spot" (English), "gray bark," and "cane rust."

History and Geographic Distribution. Some uncertainty exists as to the original home of anthracnose. It was reported from Italy in 1879 and from America in 1882. The imperfect stage of the fungus was not described until 1877 (from *Rubus chamaemorus* in Italy). McAlpine reported it as occurring in Australia in 1897; thus its extensive distribution must have occurred much before it was first called to the attention of botanists.

Anthracnose has been reported as occurring wherever raspberries are grown. Since the native black raspberries of America are quite susceptible, it has received more attention in America than elsewhere. As late as 1906 it had not been reported in England, but by 1925 it was considered a destructive disease of the European red raspberry varieties commonly grown in southern England.

Economic Importance. If left unchecked anthracnose may cause an almost total loss of the crop in a season when conditions are favorable to the disease. Loss results from (1) defoliation, (2) reduction in size of fruit, and (3) killing of canes, either directly or as the result of winter injury following weakening through defoliation and cane infections. In some seasons some of the carpels of the berries are infected, with the result that they dry up and the whole berry is worthless. On weakened fruiting canes the crop may be reduced by 30 to 40 per cent by weight even in a year when the disease is not especially severe. The disease is of most economic importance on the back raspberry, but may cause extensive damage on susceptible red varieties such as the Latham.

Damage to blackberries and dewberries may occur in seasons favorable to the disease, but ordinarily these berries are not seriously affected.

Symptoms. The disease can be found on all parts of the plant except the roots.

On Canes. The disease first appears in the spring on the young shoots as small, purplish, slightly raised spots. As the cane grows, the spots enlarge and become lens-shaped, or oval, with a slightly raised purple edge. The central portion gradually takes on a grayish color and becomes sunken and fissured. The lesions are often so close together that they form large irregular areas with ill-defined edges. The central area on the individual spots turns to a grayish white (Fig. 63). Late infections (August and September) result in another type of lesion. These are especially noticeable on red raspberry canes. They appear as superficial, gray, oval areas with no pitting. The individual spot is definite in outline, but the lesions are often so numerous as to cover sections of the entire cane, giving it a distinct light gray appearance. This is the so-called "gray bark" phase of the disease (Fig. 64). On both types of lesions small dark dots may be observed. These are especially noticeable in the gray-bark lesions, where they often have a radiating appearance. During the early part of the second year the cracks which developed during the previous summer are deepened, and the canes are further weakened by the loss of the underlying tissues.

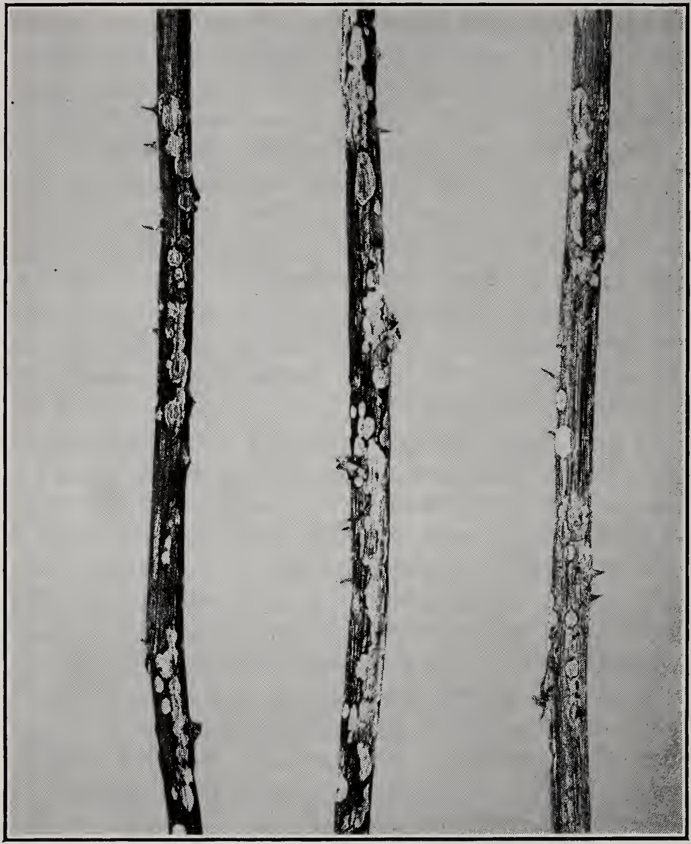

Fig. 63. Raspberry anthracnose on canes of black raspberry.

On Leaves. On the petioles and larger veins of the leaves the lesions are similar in general appearance to those on the cane but are elongated. Leaf spotting on the blades of the leaflets first appears as small, indefinite, purplish areas which later enlarge and turn brown. Anthracnose leaf spots should not be confused with *Septoria* leaf spots which are more definite in outline and have a distinct white center.

On Inflorescence. Pedicel lesions resemble those on the petiole of the leaf and sometimes cause a withering of the fruit while still in the green stage. Lesions on the *calyx* are quite common but rather difficult to see because of the hairy coating of this organ. The result of the attack is seen later as a wilting and drying of the tips of the calyx lobes. Infection on the *fruit* itself is not common, but when present appears on the individual drupelets which become shrunken and turn brown. If infection is severe on the fruit, the whole berry appears dry and "seedy" when ripe.

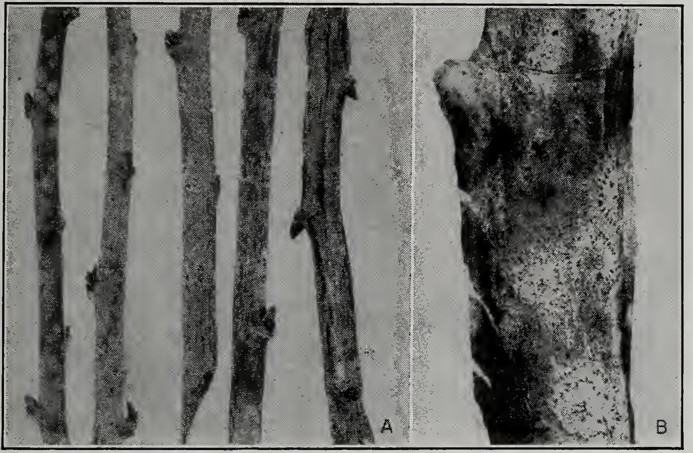

Fig. 64. Raspberry anthracnose. Gray bark stage on Latham, showing light and heavy infections and bark cracking. At right enlarged to show acervuli in individual lesions.

On blackberry leaves the spots are more indefinite and darker in color than on the raspberry.

Some varieties of dewberries are much more susceptible than blackberries, and often the fruit pedicel is girdled, resulting in withering of the fruit before maturity. The symptoms on the other organs are similar to those on black raspberries.

Causal Organism. Anthracnose is caused by a fungus *Elsinoë veneta* (Burk) Jenk.[1] The imperfect form, *Gloeosporium venetum*, was described by Spegazzini in 1877 in Italy on *Rubus chamaemorus*. The fungus on the raspberry in America was named *G. necator* by Ellis and Everhardt in 1887, but the next year Scribner pointed out the identity of the two descriptions which Ellis had originally acknowledged, and *G. venetum* Speg. was retained for the fungus in America.

In 1914 Burkholder reported the discovery of the perfect stage of the fungus and named it *Plectodiscella veneta* in 1917, after showing the connection between the two forms. The generic name of the fungus was changed to *Elsinoë* by Jenkins in 1932, and she considered the imperfect stage a *Sphaceloma* rather than a *Gloeosporium.*

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[1] Class Ascomycetes, Order Myrangiales, Family Myrangiaceae.

Morphology. The slow growth of the fungus results in beaded hyaline *hyphal threads* so compact as to form a stromatic pseudoparenchymatous tissue beneath the cuticle of the host. Short unbranched conidiophores arise from this stromatic pad in acervuli. The *conidia* are hyaline, continuous, oblong to elliptical, and measure 5 to 7 by 2.5 to 3 μ.

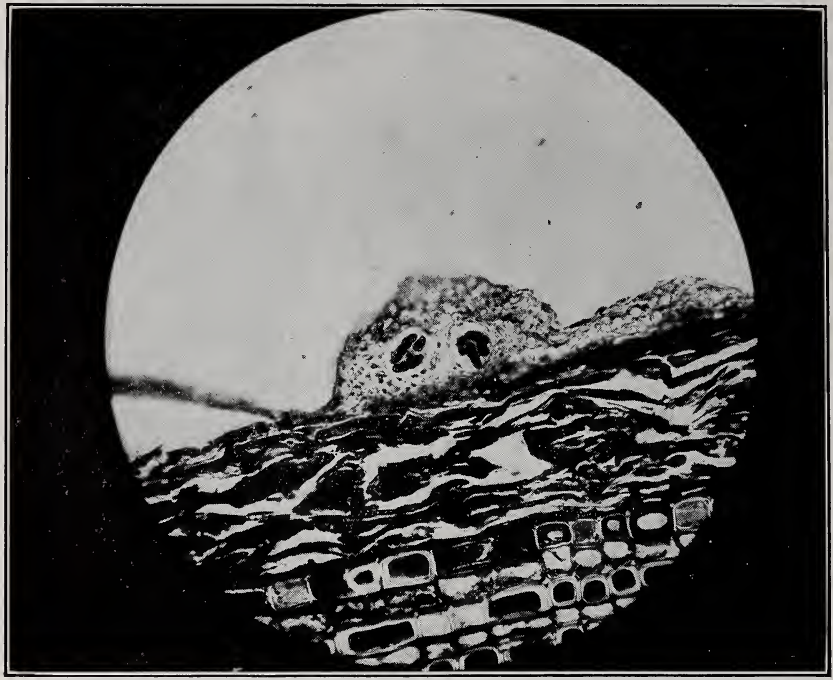

FIG. 65. Section of stroma of *Elsinoë veneta* showing embedded ascus and ascospores.

The *ascocarps* are borne on a stromatic cushion and are about 75 μ in diameter. The *asci* are formed in the interior of the ascocarp. They consist of globose thick-walled cells from 24 to 30 μ in diameter, scattered and separated by the fungus tissue of the fruiting body (Fig. 65). In this globose ascus the eight hyaline, four-celled, ovate, slightly curved *ascospores* are borne in a parallel arrangement. They measure from 18 to 21 by 6.5 to 8 μ.

Disease Cycle. In the lesions on the canes formed the previous season, both conidia and ascospores are produced early in the spring and infect any exposed new growth. It is not clear as to the relative importance of these two types of spores in bringing about primary infection. Harris, in England, is of the opinion that much of the early infection is caused by the ascospores, while in America most investigators have had difficulty in finding the perfect stage of the fungus and thus regard the ascospores as of no great importance. The conidia may be found in abundance quite early in the spring and could thus account for the infection as soon as growth starts.

The ascospores are forcibly discharged from the ascocarp during damp periods, and when alighting on the surface of exposed parts become firmly attached. They germinate by a yeastlike budding process, and these buds round off into typical conidia. These in turn germinate in the usual manner and infect the host tissue.

The conidia formed in typical acervuli are washed from the old lesions during rainy periods, become firmly attached to the developing plant parts, and germinate in from 3 to 12 hours. The germ tube penetrates the cuticle of the tender stem, leaf, or flower pedicel. After penetration the fungus mycelium spreads between the cuticle and epidermal cells. Later the epidermal cells collapse, and the fungus evidently produces a toxic substance which first stimulates rapid division of the subepidermal parenchyma cells. This causes elevation of the young lesion above the surrounding healthy tissue, but the parenchyma cells later collapse, so that the lesions become sunken as growth of the healthy tissue continues. The mycelium continues its growth inward through the parenchyma and phloem to the cambium and even into the xylem (wood) of the cane. In the meantime a thin stroma fills the cavity of the lesion, and on this new acervuli are formed.

Infection apparently is confined to the young actively growing parts of the plant. The numerous lesions at the base of the cane are the result of infections which occur shortly after the shoot emerges from the soil. The fungus in the lesions from which the primary infection occurs, that is, on the fruiting canes, soon ceases to produce spores and evidently dies out during the summer. On new canes two types of lesions may be observed in late summer, the first type as described and a superficial, or "gray bark," type. The superficial lesions are especially abundant on red raspberry canes, and it is from the numerous fruiting bodies on these lesions that both conidia and ascospores are produced for the spring infection. These lesions, as the name implies, are quite superficial and do not extend into the bark more than a few cells. The fruiting bodies in this type of canker are arranged in a rather definite radiating manner quite different from those on the pit-type lesion. Just when the infection occurs which gives rise to the superficial lesions is uncertain. Since these lesions are produced on fairly mature bark, it would appear that the failure to penetrate is due to the maturity of the bark tissues and that the fungus develops more or less as a saprophyte.

The ascocarps are produced on the stroma late in the summer, and the asci are formed in the fall, but the ascospores are not matured until early spring. The lesions formed on the new canes the previous season also produce acervuli in abundance at this time, thus completing the life cycle.

Control. Spraying is the most widely applicable method of controlling anthracnose, although supplementary measures such as sanitation and selection of resistant varieties are of importance.

The most important sprays are those applied early in the season for the prevention of primary infection. The delayed dormant spray of a strong Bordeaux mixture or lime sulfur may act as an eradicant spray as well as a protectant. Another application is made to protect the developing shoots just prior to blooming. Ferbam at 2 lb to 100 gal is recommended for this application, and a wetting agent should be added. Another ferbam application immediately after bloom is advisable where trouble has been experienced in controlling the disease in previous years.

Black raspberry plants are received from the nursery with a portion of the runner stem attached for convenience in handling. These "handles" often show anthracnose lesions and should be removed before setting the plants.

Quillen, a black raspberry variety of good quality, is reputed to be quite resistant to anthracnose.

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Verticillium Wilt of Brambles

Various common names have been applied to diseases of many crops caused by *Verticillium*. On brambles the terms "blue stem," "western blue stem," and "blue stripe wilt" have been used. These names are descriptive for the disease as it occurs on black and red raspberries, but have not been adopted by American authors since a virus disease was also called blue stem and on some brambles the bluish condition of the cane is not a striking symptom or is entirely lacking.

History. The earliest record of a *Verticillium* causing a plant disease was by Reinke and Berthold in 1879 who isolated the organism from diseased potatoes and named it *Verticillium albo-atrum*. The earliest record of *Verticillium* on *Rubus* species was by Lawrence (1904) in the Puget Sound region where it was causing serious trouble on black raspberries. In 1912 he named the fungus *Acrostagalagamus caulophagus*, which is now considered a synonym of *V. albo-atrum*. Harris reported it on red raspberries in England in 1923 and called the disease "blue stripe." In 1926 Berkeley and Jackson reported on a disease of red raspberries in New York and Ontario, Canada, caused by a *Verticillium* which they named *V. ovatum*.

Information on the origin of the disease on bramble crops is lacking. The soil organism *V. albo-atrum* is very extensively distributed and parasitizes a very large number of plants, such as eggplant, potato, and tomato. Thus it is probable that it became prevalent in the soil where such plants were grown, and when these areas were planted to raspberries the disease appeared.

Rudolph in 1931 published the results of an extensive investigation on *Verticillium* hadromycosis, in which he called attention to the extremely wide range of plants subject to attack by *V. albo-atrum*. In this publication he briefly describes the disease on blackberry, dewberry, youngberry, and red and black raspberries.

Geographic Distribution. *Verticillium* wilt on brambles has been reported from most sections of the world where raspberries are grown and where conditions are favorable for the development of the fungus. The disease is favored by cool conditions and consequently is most frequently found on brambles in the northern sections of bramble-growing regions. For example, in the United States it is rarely found below the 35° latitude, but occurs south of this latitude along the Pacific Coast.

Symptoms. On black raspberry the earliest sign of the disease is noticed on the current year's *canes* in late spring. The lower leaves show a dull green cast as compared to the bright green of normal leaves. Only a few of the young shoots may show symptoms. The disease progresses up the stem, causing yellowing of the older leaves and finally defoliation. In this process the shoot is stunted by a shortening of the internodes. Wilting is usually first evident at the tip of the shoot, since the fungus in the stem cuts off the flow of water to this actively developing terminal portion. Finally the entire shoot withers and dies. The symptom giving rise to the term blue stem is evident on the shoots of most raspberry varieties and may consist of a dark blue color entirely over the stem or in stripes. In an advanced stage of the disease the vascular system is discolored and has a water-soaked appearance. The brown discoloration of the woody cylinder is more evident in the roots. Frequently symptoms develop only on one side of the shoot or on one or two shoots of several in a "hill." This is due to the fact that the aboveground symptoms are the result of the root infection, which may not involve all the roots of the parent crown.

The first year after infection many of the new shoots may survive. These become the fruiting canes of the second year. If these were infected the previous year, they will either be dead or will show poorly developed buds, frequently along one side of the cane. In what appears to be healthy canes the buds may open in a normal manner, but soon the expanding leaves are stunted and show a yellowish color. These clusters of leaves later wither, together with the laterals to which they are attached. In some cases the effect develops late enough so that fruit is formed, in which case the berries are small and insipid.

The symptoms on red raspberries are in general the same as on the blacks, but since the new canes are produced at a distance from the parent plant, these are often free of the disease even where the parent plant (fruiting cane) shows signs of infection. It has been observed that the amount of suckering is reduced in red raspberries, thus giving the appearance of a thin stand.

Economic Importance. The main loss is the death of bearing plants in a field. Some loss results when infected plants are not killed for several years, but are stunted, and produce only a few berries on weakened laterals. The percentage of infection is usually very high when raspberries are planted on land in which solonaceous crops such as potato and eggplant had been grown previously, even where these crops showed little wilt. In Oregon, for example, in an acre of 3-year-old Plum Farmer black raspberries 52 per cent of the plants were either killed or were dying. In regions where raspberries are continuously grown the disease becomes progressively worse. On red raspberries the loss is usually much less since sucker plants often escape the disease, and thus fruit production may be sustained for several years even where the original plants were diseased.

Causal Organism. The fungus causing *Verticillium* wilt is *V. albo-atrum* Reinke and Berthold.[1] This is an extremely variable fungus, and mycologists often differ as to species classification. Entirely different cultural characters often appear in cultures from a single source, or sectors may develop in a monosporic culture. There are also strains varying in pathogenicity on certain hosts. It is generally agreed that the wilt organism on brambles is the same as that originally described from potatoes, although several authors have suggested that it might be considered a separate species.

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[1] Class Fungi Imperfecti, Order Moniliales, Family Moniliaceae.

V. albo-atrum produces a septate *mycelium*, hyaline when young but later producing dark-colored hyphae. Small dark *sclerotia* are commonly formed in culture, on the host plant and in the soil. When exposed to moist conditions the hyphae produce numerous *conidiophores* with whorled branches, on the tips of which are borne the ovoid, hyaline, continuous *conidia*.

Hosts. *V. albo-atrum* has been reported from several hundred different host plants, both woody and herbaceous. It is a common parasite on coniferous plants. Recently it has been reported as causing serious damage on strawberries and seems to be one factor in the cortical root rot complex of this plant (see page 420). It is very common and destructive on solanaceous crops such as potato, tomato, eggplant, and okra.

Disease Cycle. *V. albo-atrum* is a soil-inhabiting fungus which can live indefinitely in the soil either as mycelium or sclerotia. It is probable that most infection of the roots of brambles results from the mycelium rather than from conidia. The conidia, however, are important in the dissemination of the fungus, since when produced on the aboveground organs they may be wind- or water-borne. Penetration is usually through the roots, but stem infection at or near the ground line may occur. The mode of infection has not been definitely determined, but the fact that plants such as eggplant can be readily infected by merely adding the fungus to the soil indicates that wounding is not necessary. After penetrating, the fungus enters the vascular system and, by its growth and production of a gummy material, interferes with the uptake of water, thus bringing about wilting. Later the phloem is also invaded. There is evidence that the fungus travels rather slowly through the undisturbed soil but that it can be distributed by cultivation and water currents. However, Zeller reports that at the end of one year healthy plants were 68 per cent infected when planted at a distance of 30 in. from diseased plants, and at the end of the second year 9 per cent were infected at a distance of 90 in.

Temperature is by far the most important factor involved in the severity of the disease. That it is a cool-weather fungus has been demonstrated both by controlled experiments and by the geographic range of the disease.

Many observations and experiments have demonstrated that *Verticillium* wilt of brambles is influenced by the type of crop plants previously raised on the land. Eggplants, potatoes, and tomatoes are especially susceptible and frequently are responsible for heavy losses in brambles following these crops.

Control. Since *V. albo-atrum* is a soil-inhabiting fungus and infects mainly through the roots, obviously it cannot be controlled by the use of protective sprays.

Crop rotation of 3 or 4 years using mainly grain crops has been found to reduce infection on brambles when planted on land previously infested. It is always inadvisable to plant brambles on land following solanaceous crops (eggplants, tomatoes, or potatoes), unless it is certain that these crops were free of the disease.

Most commercial varieties of black raspberries are susceptible. Among the red raspberries Zeller claims that Cuthbert is highly resistant under Oregon conditions, but Fulton found this variety susceptible under Michigan conditions. Thus at present resistant varieties of either red or black raspberries are not available. Blackberries and dewberries rarely suffer much from wilt, although they are known to be susceptible.

Plants for new plantings should be obtained from nursery plantings known to be free of the disease.

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Orange Rust of Brambles

The bright orange color of infected leaves on blackberries is responsible for the one common name which has been applied to this disease.

History and Geographic Distribution. The early history of this disease can only be surmised from the reports of mycologists who named the fungus. It was described from Kamchatka in Asia in 1820 and from eastern North America in 1822, and its early-known wide distribution in the North Temperate Zone on a number of *Rubus* species indicates that the disease had been present long before it was scientifically described.

In North America it is found from central Alaska to southern California on the West Coast and from southern Canada to Florida in the east and westward to the dry Great Plains area. It has not been reported from the South Temperate Zone.

Symptoms. The disease can be identified shortly after new growth appears in the spring. The slender infected *shoots* develop a lighter green foliage than uninfected plants. The *young* leaves even before they unfold are dotted with small, glandular, yellowish bodies, which later turn black. Two or three weeks later the lower surface of the leaves is covered with a bright-orange-colored mass of spores, at first waxy but later powdery in texture (Fig. 66). These sori commonly extend over the whole undersurface of the leaflets, but may be only on a limited area. Usually most of the leaves on a shoot show the disease. Shoots arising from infected roots in the spring are slender and often bunched, that is, several instead of the normal one arising from the underground bud. Buds on old infected canes also send out several lateral shoots in the spring, thus giving a witches'-broom effect to the bearing cane. The plants may become decidedly dwarfed, but may remain alive for several years.

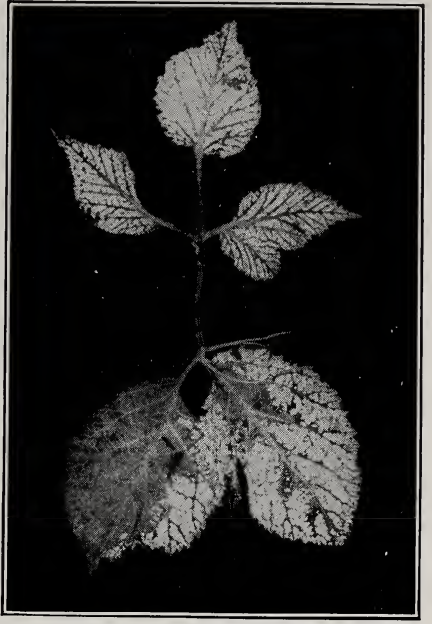

Fig. 66. Orange rust of brambles, showing orange-colored spore masses covering most of lower surface of blackberry leaf.

Economic Importance. An infected plant is a total loss as a producer. Since the fungus is perennial in the stem and roots, all new plants developed from the original infected plant will be rusted.

Causal Organism. The fungus *Gymnoconia interstitialis* (Schl.) Lag.[1] is an autoecious species; that is, it completes its life cycle on the one host. When a short-cycle form of this rust was discovered, Arthur erected a new genus, *Kunkelia,* for this type and suggested that the demicyclic form in which true teliospores are produced be named *G. peckiana* (Howe) Trotter, but it is now generally accepted that the short- and long-cycle forms are so closely related that genus or species separation is not warranted. Both forms give much the same symptoms, and their geographic distribution is much the same, although the short-cycle form is likely to have a more southern range. Kunkel also found that in the vicinity of Washington the rust on black raspberries was always of the long-cycle form, while the short-cycle form was more common on blackberries and dewberries.

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[1] Class Basidiomycetes, Order Uredinales, Family Pucciniaceae.

The small glandular spots which appear early are the *pycnia*. They are mostly on the upper leaf surface and originate from the subepidermal tissue of the leaf.

In both forms the mass of spores seen on the lower surface arise in pustules which correspond to an *aecium*, but without a distinct *peridium*. The aeciospores are produced in chains from a subepidermal *hymenium*. The aeciospores vary somewhat as to size in the two forms, the short-cycle one having the smaller spores. The aeciospores, orange-colored in mass, are globoid, 16 to 24 by 19 to 30 μ, with finely verrucose uncolored walls. In the short-cycle form these aeciospores function as teliospores, producing a four-celled *basidium*, each cell of which gives rise to a *basidiospore*. In the long-cycle form the aeciospores on germination form a slender germ tube, but in rare instances may produce a basidium. In the long-cycle form, true teliospores are formed. The telia appear on the underside of the older leaves as small dark brown pulverulent spots. The *teliospores* are two-celled, smooth, and measure 18 to 27 by 32 to 45 μ. The telia are often overlooked because of their appearance before the aeciospores are mature. It is probable that they play little part in the disease cycle. The uredinial stage is lacking in both forms.

Hosts. Most species of *Rubus* are more or less susceptible, but the red raspberry is not known to be susceptible. Rust is especially prevalent on wild and cultivated blackberries and dewberries in northern United States, but is rarely found on black raspberries in this region, although it is said to be common in the South. Orange rust is known to occur only on *Rubus* species.

Disease Cycle. Infection may originate from either aeciospores or from the basidiospores arising from the aeciospores or from teliospores. Pady in 1935 demonstrated that contrary to the general conception, penetration by the germ tube was not always through the stomata. He found that the germ tube on coming in contact with the leaf surface produced an appressorium-like structure, and from this an infection "peg" penetrated the cuticle, and the penetrating hypha invaded the epidermal cell. From these cells the hyphae pass into the intercellular spaces in the subepidermal layers. The mycelium grows rapidly through the intercellular spaces of the mesophyll, and where it comes in contact with the cells, haustoria are formed within the host cells. That infection can take place through the cuticle is further evidenced by the fact that inoculation on the upper surface of the leaf where few if any stomata are present will bring about infection.

The young new blackberry shoots arising from the crown or from the root runners are infected by the sporidia or aeciospores as soon as they are dispersed from previously infected plants. The fungus develops in these shoots, growing into the stem tissues and into the pith. The hyphae invade the underground portions of the plant, and as the runner roots develop, the fungus extends into these, so that the plants arising from them the following spring are invaded. The initially infected plant may develop in a normal manner without evidence of the disease during the remainder of the season. When it renews growth the following spring the typical symptoms will appear. The new plants arising from the infected root system will also show the disease. In the black raspberry, infection of the cane tips that are in the process of rooting takes place in late summer, and thus the disease appears the following year.

Jarring an infected plant gives rise to clouds of spores which are easily carried by currents of air for considerable distances. The yellow coating of spores clinging to one's clothing after brushing diseased plants is evidence of the millions of spores carried along by any moving object. Under these circumstances one would expect epidemics of this disease where a few infected plants are present. As a matter of fact, it is rare to find extensive infection on groups of wild or cultivated brambles. While no thorough study of the climatic conditions favoring infection is available, it is probable that a sequence of such favorable conditions must prevail in order for extensive infection to occur.

Control. Selection of resistant varieties of blackberries is of importance where such varieties are adapted to the region concerned and have desirable market qualities. For example, Eldorado grown for many years in the region where both wild and cultivated blackberries are heavily rusted is rarely infected. In addition to this variety, Oregon Evergreen, Russel, and Snyder are resistant. Ebony King, a newly introduced variety, is reported as resistant. Alfred, Crandall, and Young are susceptible. Boysenberry is moderately resistant, and Leucretia dewberry is reported as resistant. Red raspberries seem to be immune, but there is only limited information concerning resistance in black raspberry varieties.

The orange rust fungus is systemic and perennial in the host; thus a plant once infected carries the fungus throughout its life and all plants propagated from it will be diseased. Plants for setting new plantings should be obtained from areas where the disease is not present. Since the rust does not show on the infected plants the first year, there is always the chance that the disease will show up later in transplants, but at this stage of growth they can be easily rogued.

When rusted plants are found in an established planting, they should be dug up and all roots removed from the soil. Since the diseased plants can be detected before the aeciospores are mature, it is possible to remove these without dispersing the spores. Wild brambles in the neighborhood of cultivated plantings should be eradicated. Chemical weed killers may be used for this purpose.

Protective sprays are of doubtful practical value for the control of orange rust alone, but no doubt sprays applied for the control of other diseases may aid in rust control.

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Other Rusts of Brambles

Late leaf rust caused by a heteroecious rust [*Pucciniastrum americanum* (Farl.) Arth.] may cause serious damage to susceptible varieties of red raspberry, but since it usually appears late in the season and only occasionally in a severe form, it may be considered a minor disease. This rust does not occur on black raspberries or blackberries.

The wild red raspberry *Rubus strigosus* in the eastern United States is very susceptible to this rust, and a number of cultivated varieties originating from this species, such as Cuthbert, seemingly inherit this susceptibility. Latham has been found to be very susceptible under southern Illinois conditions. While this rust occurs throughout the northern half of the United States and southern Canada, it is more common east of the Mississippi River. It has not been reported from other countries.

Diseased leaves are covered with fine light yellow powdery masses of spores which appear on the older (basal) *leaves* in midsummer. These spore masses are seen also on the *leaf petioles*, the *shoots*, on the *calyces* of the fruit, and even on the *fruit* itself. While the leaves are not killed, the effect of the attack is to cause curling of the leaflets and, later, defoliation.

When the rust is present in a severe form the canes are bare by September, except for a few curled leaves at the tips of the canes.

The rust fungus produces the *uredial* and *telial* stages only on the raspberry. The alternate host is spruce (*Picea canadensis*), on which the *pycnia* and *aecia* are produced. However, it is certain that the rust on raspberry may be independent of this source of inoculum since the disease is found year after year in regions remote from any spruce trees. It is probable that the fungus lives over in the raspberry canes and the following growing season produces the urediospores responsible for the renewal of infection.

The small, numerous light yellow spots seen on the undersurfaces of the leaves are the *uredial pustules* of the rust. The *urediospores* are borne at the apex of a cone-shaped structure which protrudes above the epidermis. These spores, as is usual in the rusts, are able to bring about a continuous infection over the remainder of the season. The black, one-celled *teliospores* are found later in the season intermingled with the uredial pustules. They are capable of infecting spruce through the production of sporidia, as proved by inoculation, but probably play little part in the life cycle of the rust on *Rubus.*

Control. The relative unimportance of this disease so far as production is concerned hardly warrants special control measures.

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Dodge, B. O.: Morphology and host relations of *Pucciniastrum americanum. J. Agr. Research,* **24**:885–894 (1923).

Blackberry yellow rust [*Kuehneola uredinis* (Link.) Arth.] in symptoms is similar to the western yellow rust of raspberries. It is common only on blackberry and dewberry in eastern North America, although it has been reported on a number of species of *Rubus.* It is also present in Europe. In 1950 Fisher and Johnson published on this rust as it occurs in the Pacific northwest. Their investigations cleared up a number of points in the life cycle of the fungus and host relationships. They found it caused a serious disease of the cultivated Evergreen blackberry, *R. laciniatus.* In addition, it was prevalent on the Pacific Coast dewberry, *R. macropetalus.* The *uredial* stage appears early in the spring on the *fruiting canes,* thus indicating that the fungus overwinters as uredial mycelium in these canes. The *urediospores* then infect the developing leaves on these canes, giving rise to numerous, minute, yellow *uredia* on the lower surface of the leaves. The *telia* are inconspicuous and appear as buff-colored areas in the fall among the uredia. Unlike most rusts, there is evidence that the teliospores germinate in the fall and infect the new growth at this time, since Fisher and Johnson found *pycnia* and *aecia* developing in late October and early November. These were found on the lower leaves near the ground on the canes of the current season's growth, thus indicating infection from the *sporidia* derived from the teliospores on the upper leaves of the fruiting canes or from the fallen leaves. However, since this rust fungus overwinters in the uredial stage on the fruiting canes, it is probable that neither the aeciospores or teliospores are of importance in the life cycle. The long chain of cells of the *teliospore* is a distinctive microscopic feature of this fungus.

This rust is of little economic importance since most cultivated blackberries appear to be relatively resistant and it is likely to cause injury only in regions and seasons of heavy rainfall and humid conditions. Rust was found on the fruit in Washington during an exceptionally wet season, but it is probable that little loss is ordinarily sustained. There is no doubt but that the canes are weakened by defoliation and splitting where the disease occurs in a severe form.

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Fisher, G. W., and Folke Johnson: Cane and leaf rust, *Kuehneola uredinis* (Link.) Arth. of blackberries in western Washington. *Phytopathology,* **40**:199–204 (1950).

Western yellow rust, or "cane rust," of raspberries caused by an autoecious rust *Phragmidium rubi-idaei* (DC) Karst. is of importance only in the Pacific northwest. It has been reported, however, from the Atlantic to the Pacific in North America and also from Europe, Siberia, and Japan. The common name "western yellow rust" is here used to distinguish this rust from late leaf rust (page 326), which is also often called "yellow rust." This yellow rust is primarily a disease of the red raspberry, although it has been reported as occurring on black raspberries, but not on blackberries.

The greatest economic loss from raspberry rust is from the effect of the lesions on the canes. Zeller in Oregon in 1927 stated, "Losses from rust usually occur during the second or fruiting year of the canes. The infections near the ground have become deep and cankerous by the second year. If they are numerous they not only hinder the normal rise of sap but also produce brittleness of the canes." Canes severely infected fail to mature fruit, or the fruit is undersized or withered. The fungus does not kill the leaf, but early defoliation results, especially from the aecial infection. On the whole the disease is of little economic importance, since even where it causes considerable damage in one season it may appear only in a light form in other years. The rust attacks all of the aboveground parts of the plant, but is most conspicuous on the leaves, leaf petioles, and fruiting laterals.

The first evidence of the disease in the Northwest is in May. The *pycnia* of the rust are then seen mostly on the upper surface of the leaf as small, rather inconspicuous elevations. In these same spots the *aecia* break through in a circular arrangement around the pycnia. The aeciospores in mass are orange to pale yellow. *Aeciospores* are globose to broadly ellipsoid with light yellow papillose walls. Uredial pustules develop on the underside of the leaf, the stems, and leaf petioles. The production of the urediospores on these organs is so abundant that the whole surface is covered with a yellowish powdery coating. The uredial lesions on the stem are elliptical, giving a cankerlike effect. The urediospores are broadly elliptical, 18 to 23 by 15 to 18 μ, with echinulate walls. The teliospores are produced in the same pustules from midsummer to winter on the lower side of the leaf and on the stems. The black teliospores are cylindrical and, as is characteristic of this genus, consist of several cells, in this case eight or nine. They measure 26 to 32 by 80 to 127 μ, with a blunt conical projection at the apex.

It was thought at one time that the American rust was different from a similar one in Europe which had been named *Phragmidium rubi-idaei*, and the American species was named *P. imitans* by Arthur (1910). In 1934, however, Arthur stated that a more extensive comparison of collections convinced him that the two species should be combined.

Little is known concerning the source of initial infection in the spring, but it is assumed that the sporidia from the teliospores on the fallen leaves are carried to the new growth in the spring and thus initiate infection.

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Crown Gall of Brambles

Crown gall and hairy root as they apply to tree fruits have been discussed in detail on pages 101–113. Since crown gall is one of the limiting factors in raspberry production, a brief treatment of this disease is warranted at this point. Another gall disease of brambles has long been confused with the true crown gall and is caused by a similar organism. This disease is mainly confined to the canes and consequently is called cane gall, or "cane-knot."

Banfield in 1934 reported the results of an extensive study of the crown gall organism and its pathogenicity on the red raspberry. He demonstrated that the bacteria are given off continuously from the surface of the living crown gall into the surrounding soil water and that they remain in a pathogenic state for over a year. Incipient galls overlooked by nurserymen are often responsible for introducing the organism into newly planted fields where, with the growth of the galls, the soil becomes infested. On brambles most of the galls are found either at the ground line or on the stubs of lateral roots which have been cut or broken in planting or during cultivation (Fig. 67) and in the black raspberries by breaking of the canes where they are attached to the root crown. The extensive, rather superficial root growth of brambles is conducive to wound production, so that this crop is generally regarded as being more susceptible to crown gall than tree and other bush fruits.

Control. A careful inspection should be made of nursery-grown plants to detect any small inconspicuous galls which were overlooked by the nursery inspectors. Such plants should be discarded and burned. Long lateral roots are usually cut back at the nursery when packing. These cuts should be well calloused before planting. Growers should never plant brambles in fields known to have a history of crown gall. If brambles are to be planted on land previously set to brambles, at least a two-year interval should be allowed during which the land is utilized for grain crops. Deep cultivation and other wound-inducing practices should be avoided.

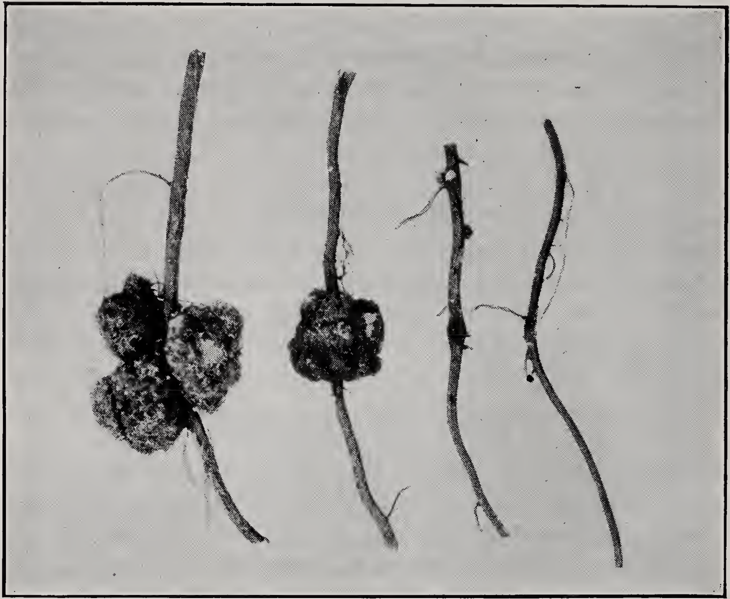

FIG. 67. Crown gall on raspberry roots. Two roots at right inoculated with cane gall organism with negative results. (*Photograph by W. M. Banfield.*)

Cane Gall of Brambles

Cane gall, also called "knot" and "aerial gall" for many years, was regarded as a type of crown gall. It was observed by Bailey on the blackberry in New York as early as 1895. It is most commonly seen on the blackcap varieties and rarely on red raspberries or blackberries. So far as known the true cane gall is not found on any host other than brambles.

Symptoms. Small spherical protuberances appear on the fruiting *canes* in late May or June. These may take the form of elongated ridges extending for several centimeters along the cane. At first the eruptions are whitish, but after rapid enlargement they become light brown in color (Fig. 68). Later in the season the surface area turns black and the spongy tissue disintegrates. The cane is often split open along the line of the infection ridges. This cracking and drying out of the canes results in yellowing of the leaves and early defoliation. The fruit on such canes usually dries up before ripening. The galls are most numerous near the ground line but may be found well up on some of the infected canes.

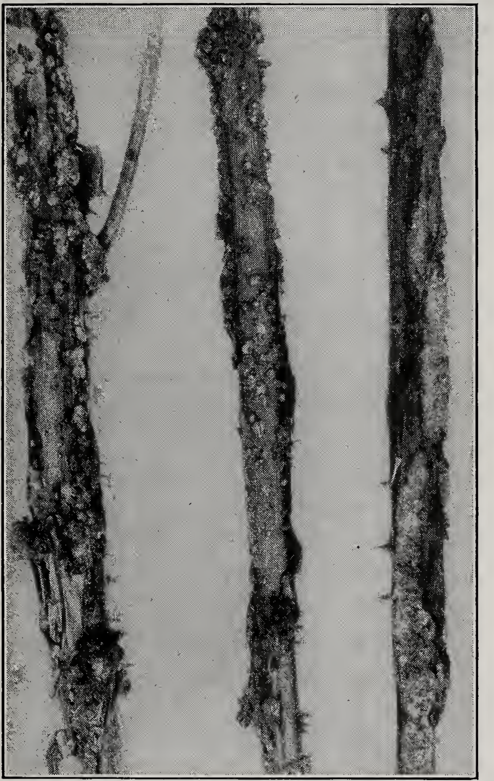

FIG. 68. Cane gall of raspberry on fruiting canes. (*Photograph by W. M. Banfield.*)

Unlike crown gall the underground parts are usually free of evident galls, although the roots are susceptible when inoculated. New shoots show no incidence of the disease during the first season of growth.

Causal Organism. Cane gall is caused by a bacterium in many respects like the true crown gall organism. Hildebrand in 1940 named it *Phytomonas rubi*, but following the present system of nomenclature it is now called *Agrobacterium rubi* (Hildebrand) Starr and Weiss. It differs from *Agr. tumefaciens* in certain physiological and cultural characters and in its pathogenicity, especially in regard to its host range and aerial rather than subterraneous development of natural galls.

Disease Cycle. Infections occur only through wounds. There is no evidence of the exact method of overwintering, although the fact that galls are not found on the current season's growth and the galls on the bearing canes appear quite early in the spring would indicate survival of the bacteria on the canes during the winter. The organism does not survive as long in the soil as does *Agr. tumefaciens*. Galls are common on the base of fruiting canes, and it is possible that the bacteria might survive the winter in this position. The bacteria are readily washed from the surface of the small incipient galls on the fruiting canes and thus infect through any wound.

Control. Rigid inspection and avoiding runner plants from infected mother plants will control the disease in most cases. Plants showing the galls should be removed as early in the spring as the galls show as a further precaution in avoiding using runners from such plants.

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See further references under Crown Gall of pomaceous fruits.

Cane Blight of Raspberries

The importance of this disease in raspberry production is questioned by some workers who regard it mainly as occurring only on plants weakened by winter injury or other agents. Others consider it of equal or greater importance than spur blight, with which it is often associated and confused.

History and Geographic Distribution. In America the disease was first observed on raspberries in New York in 1899. Since the fungus causing cane blight is very common on roses and other plants, it is probable that failure of earlier workers to report the disease on raspberries was due to lack of critical observation. Stewart and Eustace reported in 1902 on the results of inoculation experiments on red and black raspberry, using conidia from a pure culture. The disease progressed in the expected manner, and symptoms resulted, closely resembling those observed on naturally infected canes. The typical fruiting bodies and conidia were found on these canes, and no other fungus was present. They identified the fungus as *Leptothyrium fuckelii*, previously described by Saccardo from dead canes of *Rubus* and dying branches of a number of other unrelated plants.

During the next twenty years cane blight was mentioned frequently as occurring on raspberries in various parts of the United States, but no critical study was made as to the pathogenicity of the fungus, since the full account given by Stewart and Eustace was convincing.

Since the fungus causing cane blight also causes a serious disease, rose canker, on roses, it has been studied more extensively on that host than on raspberries in recent years.

In 1931 Koch in his spur blight inoculations demonstrated that the cane blight organism would cause typical cane blight symptoms in the absence of any other fungus.

Cane blight on raspberry or blackberry has been reported from many sections of North America and Europe. It is regarded as of considerable importance in England. Since the fungus causing cane blight is world-wide in its distribution, it may be reasonably assumed that the disease is present on *Rubus* species wherever conditions are favorable for its development.

Economic Importance. The loss is difficult to estimate, but it seems to be generally agreed that it is rarely very large. Bearing canes may blight back a considerable distance and yet have sufficient lower laterals to produce a fair crop of berries. Frequently weak canes of red raspberries are killed the first year, but this may be a beneficial thinning.

Symptoms. The most pronounced symptoms appear after the leaves expand on the fruiting laterals. A diseased plant will show weak growth of some or all the laterals followed by wilting of the small leaves and fruit clusters if these have started to develop. In the case of black raspberries the bark of the infected areas turns dark purple, frequently along one side of the stem only. Often infection extends downward several nodes from the cut end of topped terminals or around wounds produced by insects or mechanical injury. The invaded bark tissue turns brown later. While the early symptoms are similar to those of spur blight, cane blight is more likely to involve the whole stem and is not so definitely confined to the nodal area.

Soon after the disease makes its appearance numerous black dots may be seen on the older invaded areas. These are the pycnidia of the fungus, which later exude such quantities of dark spores that the surface of the bark has a dark gray smoky appearance (Fig. 69A). The smudgy areas on diseased canes are quite characteristic of this disease.

The symptoms on the canes during the first year of growth are usually not apparent until late in the season. If the plants have been topped or the laterals cut back, a brown infected area may extend several inches down from the wound, while uninfected canes will form a callus or die back only to the first node below the wound.

Causal Organism. The fungus causing cane blight is *Leptosphaeria coniothyrium* (Fckl.) Sacc.[1] The imperfect stage of this fungus, *Coniothyrium fuckelii,* has long been known as a semiparasitic fungus occurring on dead twigs and branches of many plants. The perithicia of the perfect stage may be found on the dead bark of the fruiting canes. They start to form in the fall, but the ascospores are not mature until spring.

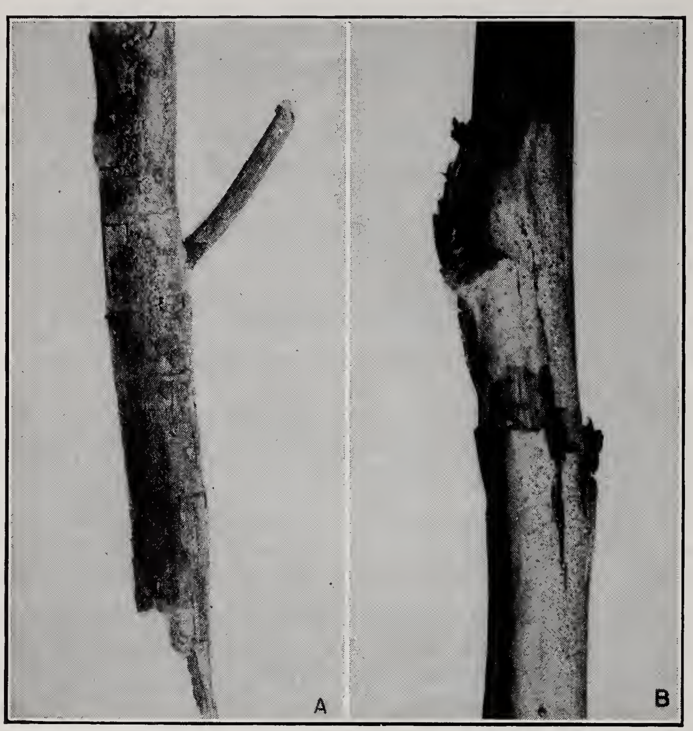

Fig. 69. Cane and spur blight of raspberry. *A*, cane blight showing pycnidia and smudges of exuding spores on 2-year-old cane; *B*, spur blight, showing scattered pycnidia, blighted shoot, and cracked bark.

The *perithecia* are subepidermal, globose, black, with a papillate ostiole. The *ascospores* are four-celled, brown, constricted at septa, oblong, and measure 10 to 15 by 3.5 to 4 μ.

The numerous *pycnidia* are superficial, dark, 180 to 200 μ; the dark *conidia* are globose to slightly elliptical, 2.4 to 5 by 2 to 3.5 μ.

Hosts. As already stated, the cane blight fungus occurs on a larger number of plants. It has been isolated frequently from soil. On economic crops it commonly causes a serious canker disease of roses and a fruit rot of apples and pears. On species of *Rubus* it is most common on raspberries, but has been reported from both blackberries and dewberries. The evergreen blackberry (*R. laciniata*) seems to be quite susceptible.

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[1] Class Ascomycetes, Order Sphaeriales, Family Pleosphoraceae.

Disease Cycle. There is some question as to whether the fungus gains entrance only through wounds, but it is generally agreed that infections are usually centered around evident wounds. Infection may occur at any time during the growing season from either the conidia or ascospores. Since the old dead canes continue to produce conidia for several years, it is probable that most initial infections are from these rather than from the more rarely formed ascospores. With the revival of growth in the spring, abundant pycnidia are formed on the fruiting canes which were infected the preceding season. Most infection occurs in late spring or toward midsummer when wounds are exposed as a result of topping new growth, insect punctures, cracking of the bark, breaking of fruit stems, etc. Koch states that he was able to infect leaves by inoculation with conidia. The canes may be killed to the ground line or so weakened as to be subject to winter injury. After gaining entrance the fungus grows rapidly through the bark, killing and browning the tissues as it advances. The limit of growth is not sharply defined except on young canes where the advance of the fungus seems to be checked by the vigor of the cane.

Dissemination of the spores may be by various agents. The very abundant exuding conidia at the tips of the fruiting canes are in a position to be easily washed onto subtending branches. The ascospores are discharged into the air and may be carried by wind currents. Insects play an important part in opening up courts of infection, and it has been demonstrated that these insects often carry loads of spores on their bodies.

Both conidia and ascospores germinate quickly under moist conditions and over a rather wide range of temperature. The fungus mycelium lives over winter in the infected canes and renews growth and spore production the following spring.

Control. Sanitation and cultural practices combined with the application of the standard fungicides and insecticides will reduce this disease to one of minor importance. The fact that the fungus lives and fruits on old canes for several years after they are removed from the field indicates the necessity of burning all such as soon as possible. Fruiting canes showing weak growth of laterals should be cut back to healthy wood. Topping should be done during clear weather so that the wounds will have a chance to dry.

Ferbam (1½:100) applied during July and August successfully controls the disease on Evergreen blackberries in Washington.

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Spur Blight of Raspberries

The disease is generally known under the common name "spur blight," but has been referred to as "cane blight," a name which is not appropriate since a disease caused by another fungus, *Leptosphaeria coniothyrium,* is properly called "cane blight." The two diseases are often associated, and both fungi may occur intermingled on the same cane.

History and Geographic Distribution. In America spur blight was first described by Detmers in 1891, but she attributed the disease to a bacterium. Peck in 1894 found the perfect stage of the fungus or spur blighted canes in New York and named it *Sphaerella rubina* Pk.

Since 1900 the disease has been reported by various writers from widely separated regions in the United States and Canada. In 1915 Sackett published an account of the disease as it occurred in Colorado, in which he confirmed Peck's observations on the presence of the perfect stage on the diseased canes. Newhall in 1923 found a *Phoma* sp. as the imperfect form of the fungus, developing on cane lesions inoculated with the ascospores of *S. rubina.* The disease was found by Zeller to be widespread in northwestern United States. Thus it may be said to be generally present throughout North America where wild or cultivated raspberries are grown. It is regarded as a more serious disease in northern and eastern sections than in the southern and middle-western areas.

The fungus causing spur blight was described in Europe in 1875 by Niessl under the name *Didymosphaeria applanata,* thus antedating the description by Peck in America. Since that date the disease has been mentioned by workers in the British Isles and in Continental Europe, indicating that it is widespread in European countries.

Economic Importance. Until recently spur blight was not considered of economic importance in North America, although in some years spur blighting and "blind" buds were mentioned as a cause of reduced production. In England and Germany considerable damage to raspberries has been reported by a number of workers. The disease appears to be on the increase in eastern United States and Canada, and in some of the New England states and around the Great Lakes region it is regarded as serious enough to warrant special control measures.

The main damage results from the blighting of spurs on the fruiting canes and killing of buds from which the fruiting laterals develop, thus materially reducing production.

Symptoms. The disease first appears on the *new canes* in late spring or early summer. Discolored areas, brown or purplish, appear on the shoot immediately below the leaf attachment. These nodal spots enlarge and extend up and down the cane for a considerable distance. The affected areas may extend from one node to another, thus involving the whole lower part of the cane, but usually are confined to an inch or so with intervening green healthy bark. The leaf arising from the node is often chlorotic in appearance, and the leaflets may fall off, leaving the petiole attached to the shoot. Dark elongated lesions sometimes are found on the midrib and larger veins of the leaflets.

On the *fruiting canes*, that is those infected the previous summer, the laterals arising from the buds in the diseased area are weak and the leaves dwarfed and yellowish. These laterals often wither and dry up early in the season. Other buds may fail to develop at all, so that the whole cane has a ragged appearance.

The infected area on the cane remains dark brown to purplish until fall, when it takes on a grayish color. Longitudinal splitting of the bark occurs in late summer, and small pimples (pycnidia) may be seen scattered over the surface of the diseased area (Fig. 69*B*). About the time the bark turns gray, numerous black pustules appear. These are the immature perithecia of the fungus. They are similar to the fruiting bodies of the anthracnose fungus in its "gray bark" stage but are more scattered and do not have the concentric arrangement characteristic of the latter.

Laterals developing from a healthy cane may be infected and show much the same symptoms, but may be killed, or so weakened that they are winterkilled.

Causal Organism. Spur blight is caused by *Didymella applanata* (Niessl) Sacc.[1] Niessl in 1875 named the fungus *D. applanata*, which was transferred by Saccardo to *Didymella* in 1882. In America it was first named *Sphaerella rubina* by Peck in 1894 and later transferred to *Mycosphaerella*. Until 1931 *M. rubina* was accepted as the correct name, but in that year Koch called attention to the paraphyses in the perithecium, thus indicating that it did not belong in the aparaphysate genus *Mycosphaerella*. By comparison with European specimens he established the fact that the earlier named *D. applanata* was the same as the American species.

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[1] Class Ascomycetes, Order Myriangiales, Family Pseudosphaeriaceae (Gäuman classification).

The imperfect stage is a *Phoma*, as determined by Newhall and confirmed by Koch. No species name has been given this *Phoma*.

Hosts. The fungus has been reported only from *Rubus* species. The disease is more commonly found on red than black raspberries, and blackberries appear to be immune. Koch inoculated some 22 varieties of raspberries and found all susceptible. Of these the two varieties of blacks, Plum Farmer and a wild form, were moderately resistant. Columbian, a purple variety, was also moderately resistant. He regarded Brighton, Herbert, and Erskine Park of the red varieties as being highly susceptible. In Illinois, Cuthbert and Latham are especially susceptible.

Morphology and Disease Cycle. The immature *perithecia* are formed in the grayish lesions in the fall, but the ascospores are not mature until late spring of the following year. The *asci* are cylindric-clavate 60 to 70 by 10 to 12 μ. When the perithecium is crushed the exuded asci are clumped (fasciculate), appearing like the "hands" of bananas. Paraphyses, usually inconspicuous, extend above the asci in the young perithecium. The hyaline, two-celled *ascospores* are ovate, with a definite constriction at the septum, the upper cell being larger and more pointed than the lower. The ascospores average about 16.5 by 9 μ.

The *pycnidia* are separate, borne free in the substratum, osteolate, not beaked, averaging about 200 μ in diameter. The *pycnospores* are hyaline to light green in color, mostly two-guttulate, elliptical to oval, 5 to 11 by 2 to 4 μ, averaging about 7 by 3 μ. Under moist conditions the pycnospores ooze through the ostiole to form a grayish white hemispherical mass.

Ascospores are matured in the spring and start discharging in late spring when the new shoots are 15 to 20 in. high. Discharge may continue through July and August. The discharged ascospores are carried by wind currents or washing to the developing plant parts where they germinate under moist conditions.

The germ tube is able to penetrate directly through unwounded tissue. After penetration the hyphae progress through the layer of cortical cells exterior to the cork layer. The cork layer seems to form a barrier, and further penetration occurs only through cracks in this layer. Cortical invasion results in the browning and final death of this tissue.

The frequent origin of the lesions at the nodes around the buds is explained by the fact that the spores alighting in the axils of the leaves and on the bud scales are in a favorable location for germination and infection because of moisture retention. Internodal infection may occur but is rare, probably because of the lack of water retention on the surface of the canes.

Infection may result from pycnospore inoculation. The pycnidia often ooze quantities of spores when under moist conditions, and these spores, washed to subtending plant parts, are capable of spreading the disease.

Control. In most areas spur blight is not destructive enough to warrant expensive control measures. Certain cultural practices such as having the plants well spaced in the row and increasing the width between the rows will aid by allowing quick drying of the foliage and canes.

Fair control by spraying has been obtained in Canada where a 3-6-40 Bordeaux mixture with a spreader was applied, when the new shoots were 6 to 10 in. in height. It is probable that the regular sprays applied for anthracnose will give sufficient control.

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Septoria Leaf Spot of Brambles

A leafspot of raspberries, blackberries, and dewberries, differing from that caused by the anthracnose fungus, is responsible for some damage to these hosts, although it is considered of minor importance. The common name "*Septoria* leaf spot" is somewhat misleading, since two closely related fungus species are associated with the disease. However, since the leaf spots are indistinguishable as to gross symptoms and have much the same disease cycle, they are treated here under the above common name.

History and Geographic Distribution. The fungus causing leaf spot was first described from Belgium about 1850. It was recorded soon after this date from North America. The disease is now known to be world-wide in its distribution.

Symptoms. Both the fungi responsible for leaf spot produce similar symptoms, but the symptoms may vary widely on the different hosts. For example, the spots on the black raspberry are distinctly white when mature, while those on blackberries and dewberries retain a light brown tinge, although some of the old spots may show small white central areas

(Figs. 70 and 71). When the spots first appear they are light brown in color but may have a purple border on some hosts. In contrast to the rather indefinite anthracnose lesions, these leaf spots are distinctly circular in outline. The individual spots usually remain small, rarely being over ⅛ in. in diameter. The white papery center often shows a few minute dark brown scattered specks which are the pycnidia of the fungus. Elliptical lesions are present on the petioles of the leaves and sometimes on the shoots. The *Septoria* leaf spot usually appears later than the spotting caused by the anthracnose fungus with which it may be confused.

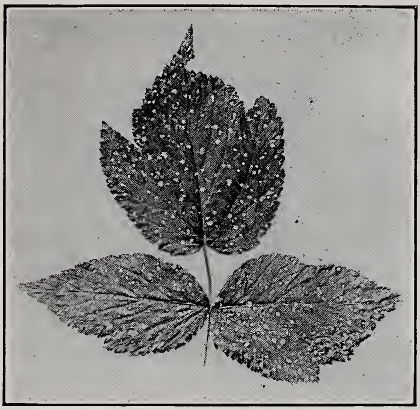

Fig. 70. *Septoria* (*Mycosphaerella*) leaf spot of brambles. On black raspberry.

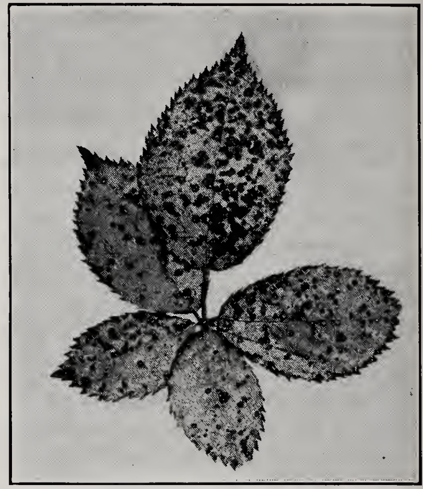

Fig. 71. *Septoria* leaf spot on blackberry.

Economic Importance. Leaf spot is of minor importance, although in some years it may cause extensive defoliation, thus exposing the berries to the hot sun. Devitalizing also renders the plants subject to winter injury. The lateness of the attack accounts for the lack of damage in most years.

Causal Organism. Two species of fungi are responsible for the leaf spotting. *Septoria rubi* Westd. was described about 1850 as the causal organism and has long been considered as the only one involved. According to Roark, two previously described species, *Ascocyta rubi* Laschin (1832) and *A. ruborum* Libert (1834) are possible synonyms, but positive identification of these has not been possible. The *Septoria* stage of the fungus on the canes was named *Rhabdospora rubi* by Ellis and Everhardt on the basis of its occurrence on the canes.

In 1917, Roark found the ascigerous stage of the *Septoria* on fallen leaves and named it *Mycosphaerella rubi*. He proved the identity of the two stages by cultural characters and inoculations. Thus the correct name of this fungus is *M.*

rubi Roark.[1] The *pycnidia* are epiphyllous, 80 to 100 μ broad by 75 to 85 μ high, brown in color, with ostiole not projecting (sometimes the pycnidia are cuplike, resembling an acervulus). *Spores* are hyaline, filiform, one- to four-septate, 20 to 55 by 1.5 to 2.5 μ. The *asci*, according to Roark, are found in the fallen leaves of several species of *Rubus*. The *perithecia* are mainly hypophyllous, usually gregarious, globose, 60 to 80 μ in diameter, with a short ostiole. The *ascospores* are hyaline, slenderly fusiform, or two equal cells, 20 to 25 by 3.5 to 4.25 μ.

Roark states that he found distinct cultural strains as regards spore production and stromatic development. This may explain the frequent variations in descriptions of leaf spots by various authors. Roark found that strains from blackberry would not infect raspberry, nor would the raspberry strains infect blackberry.

In 1937, Zeller reported that there were two distinct species of *Septoria* responsible for leaf spotting on *Rubus*. In 1916 Saccardo had noted a difference between the typical *Septoria rubi* and one occurring on *R. hispidus* collected in New York. He named the fungus *S. rubi* West. var. *brevispora* Sacc. Zeller examined specimens from a number of localities in the United States and England and came to the conclusion that the two were sufficiently different to warrant giving specific rank to Saccardo's variety *brevispora*, and so named it *S. brevispora*.[2] Darrow, in 1935, had stated that Lloyd George, Viking, and Newburg were so susceptible to leaf spot that they could not be grown commercially so far south as Maryland. Zeller found the Lloyd George, for example, was only slightly susceptible in Oregon, and on examination of the specimens from Darrow came to the conclusion that the species causing severe defoliation at Beltsville, Maryland, was *S. brevispora*, while in Oregon the typical *S. rubi* caused little damage to varieties reported as very susceptible to *S. brevispora*. From his collections he found that *S. rubi* was present on most of the material from the Pacific northwest, Wisconsin, and Durham, North Carolina, and from England. Leaves from Beltsville, Maryland, and Willard, North Carolina, showed *S. brevispora* as the causative agent. Other young dewberries from North Carolina showed both types of spots.

The main morphological differences between the two species are in the size and shape of the conidia and the fact that the ostiole of *S. brevispora* is broad with no rostrate tendencies. The perfect stage of *S. brevispora* is not known.

Hosts. *Septoria* leaf spot has been reported as occurring on a large number of species of *Rubus*. Cultivated species of red raspberries, blackberries, and dewberries are susceptible. Darrow states that in general Asiatic species are highly resistant to the leaf spot at Beltsville as compared to Latham and Chief varieties of red raspberries.

Disease Cycle. Only a limited amount of information is available as to the overwintering stage of the fungus. The fact that the perfect stage of the *Septoria* is rarely found indicates that this is not generally responsible for initial infection. The fungus lives over winter on the canes and produces pycnidia with viable spores in the spring. This indicates the probable source of spring infections. Ordinarily few spots are present on the leaves until late in the spring, but if weather conditions are favorable, there is a rapid build-up of leaf spotting throughout the summer. Oozing of pycnospores may be observed following damp weather, and washing of these to neighboring leaves accounts for the rapid spread of the disease. Infection of the new canes may take place during this period. In addition, the fungus lives over winter on the fallen leaves, producing a thick-walled pycnidium in which pycnospores are produced in the spring. Splashing of these spores on the developing foliage is responsible for some primary infection in the spring.

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[1] Class Ascomycetes, Order Sphaeriales, Family Mycosphaerellaceae.

[2] Class Fungi Imperfecti, Order Sphaeropsidales, Family Sphaeropsidaceae.

Control. The general practice of spraying for anthracnose has served to reduce the damage from this leaf spot, although the disease may build up later in the season after the anthracnose sprays have been discontinued. Fermate has been effective in control in recent experiments.

Breeding experiments by the U.S. Department of Agriculture and the North Carolina Experiment Station have resulted in the development of highly resistant varieties. Where the disease is of more than minor importance, the grower is advised to consult with these agencies for approved resistant varieties for his particular locality.

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Minor Diseases of Brambles

Rosette Disease or Double Blossom of Blackberries and Dewberries. Described by Cook in 1910 as "double blossom," this disease was further studied by Plakidas, who suggested that an older name by Norton, "Rosette," was more appropriate than "double blossom." The first record of the disease was a collection from Cobden in southern Illinois about 1884. It has since been found to be generally distributed through the southeastern and southern United States but has not been reported from the Far West. The disease is especially destructive to blackberries and dewberries in these regions. So far as known it occurs only in erect and trailing blackberries (dewberries), both cultivated and wild.

Symptoms. The disease is recognized by the rosette effect resulting from the abnormal production of multiple short bunchy shoots from the overwintering canes. The foliage in this witches'-broom is lighter in color than on normal shoots and may turn to a yellow or bronze color later. In case of severe infection all the buds on the cane may be affected, but more often only a few of the lateral branches are rosetted. The diseased shoots may be entirely vegetative or may produce more than the usual number of blossoms. These are elongated with the sepals extending as leaflike structures above the swollen lower part of the bud. The petals are larger and deeper pink than in healthy blossoms and frequently fail to unfold. The diseased flowers usually fail to produce fruit.

Hosts. The disease so far as known is limited to the blackberry species of *Rubus*. The trailing blackberries (dewberries) are especially susceptible, possibly because of their low-growing habit. Cultivated varieties of erect blackberries are considered susceptible, and the disease is found on wild species such as *R. abundiflorus* and *R. nigrobaccus*. The Himalaya blackberry is listed by Plakidas as immune, but the Brainerd with Himalaya as its maternal parent is susceptible.

Causal Organism. The fungus causing rosette is *Cercosporella rubi* (Wint.) Plak.[1] This fungus is closely related to *Ramularia*, to which genus it was assigned by Wollenweber (1917). Winter in 1885 named it *Fusisporium* (?) *rubi* from material collected by Earle at Cobden, Illinois. The short *conidiophores* are produced singly or in clusters. The *conidia* are hyaline, cylindric, tapering, straight or slightly curved, 0- to 12-septate (average 3) variable in length (13 to 96 by 2.7 to 4.7 μ), averaging 34 by 3.8 μ.

Disease Cycle. Infection takes place in the spring on the *primocanes* from spores produced on the blossoms of the fruiting canes. The spores alight on the young *buds*, germinate, and the mycelium penetrates and grows within the bud. As the infected buds grow the mycelium follows, invading the secondary or lateral buds. When flower buds begin to differentiate later, these are also invaded. The infected canes show no external evidence of the disease during the summer and fall. In the spring the development of the infected buds into the rosetted growth takes place, and when the infected blossoms open, spores are produced in abundance, chiefly on the stamens and pistils. These in turn reinfect the buds on the primocanes, thus completing the cycle. The period during which infection takes place seems to be limited to the *early* spring months, temperature probably being the limiting factor. There is some evidence that the fungus may become systemic in the crowns of dewberry plants arising from tip rootings of rosetted canes. This does not occur in upright blackberries.

Control. Prompt removal and burning of infected canes and blossom clusters in the spring before spores are produced will prevent further infection in blackberries.

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[1] Class Fungi Imperfecti, Order Moniliales, Family Moniliaceae.

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Fire Blight on Raspberries. It is well known that the fire blight organism *Erwinia amylovora* will infect a large number of species of plants, especially those of the rose family when artificially inoculated. Rarely are serious natural infections reported on hosts other than the pome fruits. An outbreak of fire blight on red raspberries occurred in Maine between 1947 and 1950. This was found to be caused by *E. amylovora,* but since cross inoculations to apple failed to produce typical fire blight, the organism was considered by Folsom to be a form species and was named *E. amylovora* f. sp. *rubi.* Later, Folsom reported evidence that the raspberry cane maggot (*Pegomya rubivora*) was responsible for the inoculation of canes by this organism.

Fire blight on raspberries has been reported from Maine, North Carolina, and Washington, but there is no evidence that it is widespread or very serious in any region. In Maine it is confined to the Latham variety.

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Virus Diseases of Brambles

Brambles, especially the raspberries, are subject to a number of virus diseases, some of which have been responsible for marked reduction in production in certain areas. The investigations of these virus diseases are of such recent date that there exists some confusion as to their exact classification and interrelation. It is well established, however, that with the possible exception of stone fruits, in no other fruit crop is the loss so extensive as a result of virus disease.

History. It was not until 1891 that a definite description is to be found in the literature of a disease which might be considered to correspond to any of the virus diseases. In that year, Detmers in Ohio described a condition ascribed by her to bacteria which closely resembles one of the mosaics now known. "Leaf curl" was mentioned a few years later by Green as being common in Minnesota. In 1902 Stewart and Eustace described a condition in New York which was known as the "Marlboro disease," to which they gave the name "yellows." They called attention to the fact that the disease probably was not of fungus origin, since it did not yield to spraying, and they suggested that it might be of the nature of peach yellows or the Bermuda lily disease, both of which were supposed to be virus diseases. Following these brief early descriptions, little attention was given to the subject until 1914, when Melchers published a more detailed account of "raspberry curl or yellows," in which he presented some exact descriptions of symptoms, together with figures which indicate that he was dealing with both leaf curl and mosaic. His statements in regard to the cause of the disease indicate that he was convinced that it was not caused by fungi or bacteria, but gave no proof that it was of virus origin, although he referred to the suggestion of Stewart and Eustace that it might be of the nature of peach yellows. It was not until the period between 1920 to 1930 that a real conception of the extent and nature of the virus-disease problem of brambles was realized. The relation of insects to the transmission of other viruses opened the way to a more definite program of research on raspberry viruses.

The first definite statement as to the method of transmission of a virus in brambles was made by Dickson in 1922. He states in his discussion of raspberry "mosaic" that "during the summer of 1920 mosaic of raspberries was transmitted by aphids." He did not determine the species of the aphids used in the experiment, "but it is highly probable that they were *Aphis rubiphila*." In 1922, Rankin and Hockey transferred "curl" from diseased to healthy Cuthbert raspberries by using *A. rubiphila*. Wilcox and Smith reported in 1924 the transfer of red raspberry mosaic to blackcaps by *Amphorophora rubi*. With the knowledge that these two aphids could be used in inoculation studies, the problem became one of determining by controlled experiments the agents responsible for transmission of each type of virus. Various workers undertook such experiments during the next ten years, the most extensive being those conducted by Bennett in Michigan between 1922 and 1928 and published in detail in 1927 and 1932.

In 1953 Folke Johnson reported "green mosaic" as being most prevalent in the northwest United States on red raspberries. "Ring spot" and "mild mottle" occurred on some varieties but were not considered of importance. "Dwarf" was the main virus disease of trailing blackberries in the Pacific Coast states and was considered of importance to the Loganberry industry. In eastern United States Jeffers in 1953 stated that "mosaic" is the most common and widespread virus disease of raspberries. He distinguishes between "green mosaic" and "yellow mosaic." Of the streak diseases he regards "mild streak" on black raspberries as more serious than "severe streak," since the latter is much more evident to the grower and can be rogued, while mild streak with its faint symptoms is likely to be overlooked, and thus the infected plants may be retained until it becomes widespread.

In the following description of the various virus diseases of brambles an attempt is made to summarize the known facts and to harmonize the various viewpoints in regard to the classification of these diseases.

In general, three main types of virus diseases are recognized, curl, mosaic, and streak. Curl, as its name indicates, is characterized by the cupping of the leaves by a downward curling of the margins (Fig. 72). *Mosaics* show various types of mottling, which in some cases are accompanied by yellowing and in others by distortion of the leaves. *Streak* is the name applied to a virus group where the chief symptom is a purple streaking of the cane.

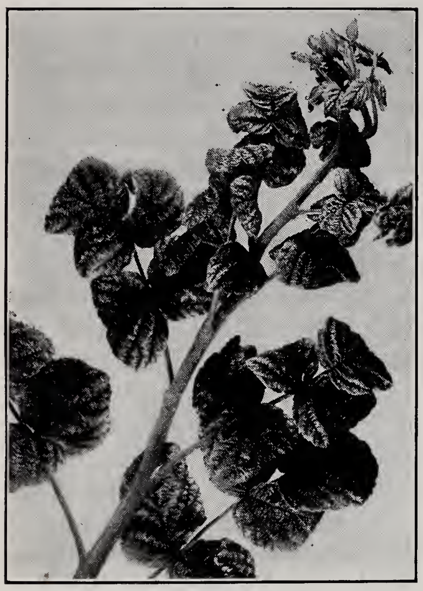

FIG. 72. Raspberry leaf curl. Shortened internodes and downward curling of leaves.

Leaf Curl. Two distinct virus diseases are characterized by decided curling of the leaves. These were named "alpha" and "beta" types by Bennett in 1930 from studies of the conditions in Michigan. Leaf curl, as it occurs on red raspberries, was described by Green in 1895, and has been recognized as common wherever the Cuthbert red raspberry is grown (Fig. 72). This common leaf curl was later named the alpha type to distinguish it from a somewhat rarer type, beta curl, which was largely confined to the Cumberland blackcaps. Bennett demonstrated that both of these types could be transmitted by *Aphis rubiphila*, but not by *Amphorophora rubi.* The beta type, while ordinarily confined to the blackcaps, will cause severe symptoms on the red variety Cuthbert and the hybrid variety Columbian.

The Cuthbert, among red varieties, is undoubtedly the most susceptible to alpha leaf curl, while Cumberland, among the blackcaps, is especially susceptible to the beta type; but the latter also causes severe curling on both the Cuthbert and the hybrid purple Columbian. Plum Farmer (black) seems to be very resistant, while Gregg is intermediate.

Transmission. Bennett conducted extensive experiments on the transmission of the leaf curl viruses by different insects between 1922 and 1928. He and others also investigated a number of methods of artificial inoculations. Successful results have been obtained only by the use of *Aphis rubiphila* as an agent. Repeated trials by Bennett and others using three other aphids and numerous other insects which feed on raspberry all failed to transmit either of the curl viruses.

A. rubiphila, the small raspberry aphis, is rather commonly found on raspberries, although the population is never large, and it does not cause noticeable direct injury. It is sluggish in its habits, which explains the localized character of infected areas in a plantation. This fact also aids in the effective roguing of the plants, without danger of extensive flights of the insect.

Unlike some other virus diseases, the period of incubation of the virus in the insect seems to be very short; in fact, the aphis is able to transmit the virus after feeding for 2 hours on an infected plant. The virus remains active in the insect over a period of at least 2 weeks, and probably throughout the life of the insect. It is not carried through the egg nor from one generation to another through viviparously produced offspring of a viruliferous parent.

Bennett has studied the movement of the curl virus in the plant and has determined that movement is confined to bark. Plants inoculated and girdled never develop the disease below the girdled portion. During the growing season, the virus is confined to the inoculated shoot and passes to the other shoots only with the renewal of growth the following season.

Unlike some of the other brambles viruses, variation in temperature does not seem to affect the symptoms of curl.

The spread of curl in a planting is usually rather slow. Often one plant will show severe symptoms, while the plants adjoining it will be healthy or show only mild symptoms. This slow spread is associated with the sluggish habits of the insect vector. High winds often carry the insects for some distance, and thus new foci of infection are established. Symptoms, resulting from the current season's infection, may appear on the tips of the new canes by the first of July.

In 1952 Cadman and Harris described a leaf curl disease of raspberries in Scotland which they considered to be etiologically distinct from North American leaf curl. This was called "yellow blotch" virus.

Control. General directions for the control of bramble virus diseases are given on page 352. Leaf curl is fairly easy to control by careful roguing. Varieties other than Cuthbert should be used when practical.

Streak (Eastern Blue Stem, Rosette). In 1923, Wilcox described a disease of black raspberries which he had been investigating for several years in Ohio. It appeared to be a virus disease, and he named it eastern blue stem because of the characteristic discolorations on the stem. Later, the more appropriate name of "streak" was adopted by most investigators. This disease was especially prevalent and destructive in a region near Cleveland, Ohio, where black raspberries were grown extensively. It has been found to be present to a limited extent throughout the states adjoining the Great Lakes and has been reported from Oregon. It is of little importance except in the northern Ohio region where it was originally discovered. It is probable that it has been widely distributed on nursery stock in the past but has not become generally established. Shortly after its discovery in Ohio, the disease was found in Michigan, Wisconsin, and Illinois, but it has never proved to be a serious menace to black raspberry production. Roguing of the diseased plants in nursery plantings in these states has, no doubt, aided in keeping the disease in check.

In 1931, Rankin described a new type of streak in western New York which he named "mild streak." He states that while severe streak is not prevalent in New York and has been recognized on only one variety, the Black Giant, mild streak, is "more or less prevalent in central and western New York."

The *symptoms of severe streak* are not pronounced during the first season. The tip leaves of the infected canes show a tendency to curve sharply downward by midsummer. More definite symptoms are seen the year following. The characteristic curling of the leaves is quite evident. This consists of a peculiar spiral twisting of the leaflets on the midrib in such a manner that the tip forms a definite hook. The leaflets themselves are bent downward and appear more closely appressed to the stem on which they are borne than is the case with normal plants. The color of the leaves is slightly darker than normal. Wilcox describes a definite mottling of the leaves, but both Rankin and Bennett state that this is by no means a constant symptom and, if present at all, is very faint.

In the second season, the diseased plants are smaller, and the fruit is inferior in quality as well as quantity. New shoots are few and weak and are easily winterkilled. From 2 to 3 years seems to be the average life of infected plants.

The most definite symptoms of this disease are seen on the canes. These may be observed to the best advantage on young shoots in early summer or midsummer. On the portion of the stem from the ground line up, dark blue or violet blue markings appear in the form of dots or longitudinal stripes of varying lengths. The stripes resemble broad pencil marks. The length and number of these markings vary from a few indistinct spots to long streaks, which may be so numerous as to give a purple appearance to the entire stem.

The *symptoms of mild streak* are much the same as for the severe type except that they are much attenuated, and the effect on the plant is not serious. The cane markings may be absent, but this is also true of severe streak in some seasons. The plants are not dwarfed in the case of mild streak. The most serious result of mild streak appears to be the effect on the fruit. The fruit seems to be halted in its development, the drupelets remaining underdeveloped with bloom showing between them.

Mosaics. The identity of the mosaics, both in America and Europe, is confused by the great variation in symptoms and the lack of definite information on the transmission of the various types. Exact classification must await the results of extensive tests which are necessary for an accurate identification and separation of viruses. The mosaics were first described by Stewart and Eustace in 1902, under the general term "yellow."

In America, two types of mosaics are now recognized: (1) "green mottle" mosaic (Red raspberry mosaic) and (2) "yellow mosaic." "Mild mosaic," referred to by Rankin and Bennett, has recently been combined with green mottle mosaic by Cooley as a result of inoculation and field tests.

In the British Isles raspberry virus diseases have been extensively studied by Harris, Cadman, and Prentice. These workers have used the patch-graft method of transmitting the viruses and thus have not had to depend on insect transmission in their attempts to resolve the various components.

Harris divides the mosaics into two classes, mosaic 1 and mosaic 2. He believes that mosaic 1 is caused by a single virus. Mosaic 2 he subdivides into "mild mosaic 2" and "severe mosaic 2." Harris in 1940 was of the opinion that mild and severe mosaic 2 were analogous to the North American green mottle mosaic. Cadman in Scotland has differentiated a number of "strains" of the yellows type of mosaic but states that these may vary under varietal and cultural conditions and for all practical purposes should be grouped under the one term "yellows."

The exact relation between the mosaic types in the British Isles and those in North America has not yet been determined.

Green Mottle Mosaic ("Red Raspberry Mosaic" and "Mild Mosaic"). Green mottle mosaic is perhaps the oldest of the bramble virus diseases and has the widest distribution. In the literature on virus diseases, it has had various names, such as "yellows," "red raspberry mosaic," "red mosaic," "mild mosaic," "curl," and "mosaic." Many of these terms were inclusive, in that all the mosaic types were grouped under the one name. Much controversy has occurred over this virus complex. Bennett selected three types which he called "mild," "medium," and "severe." He was able to transmit each type under identical environmental conditions and to show that each retained its identity over a period of 3 years, so far as symptoms were concerned. He admitted, however, that gradations of symptoms occur under varying conditions, so that it was difficult to separate the types in nature.

Green mottle mosaic occurs on both red and black raspberries, on hybrids, and on dewberry and blackberry. In Michigan it is regarded as of primary importance on red raspberries, while in New York it was considered by Rankin as the most serious virus disease of both black and red raspberries.

The *symptoms* of green mottle vary so greatly under different environmental conditions and in different varieties that it is impossible to give a general description which will fit all conditions. In some varieties and under some conditions, the symptoms may be entirely masked. As expressed by Cooley (1936): "Argument is possible over this question because the said range of symptom extremes extends from a very light mottling, visible only early in the season, and accompanied by no appreciable injury, *through all gradations*, to a 'blistered' mottling associated with severe necrotic, growth stunting, and rapid-killing effects. Complicating factors, such as weather, plant vigor, three species of hosts with numerous varieties, and possible association with the yellow mosaic virus, all affect symptom expression, and thus increase the chances for confusion and contradictory opinions as to causal relationships."

In general, green mottle mosaic is characterized by the appearance on the leaflets of mottled areas which are *darker green* than the surrounding tissue. This may be accompanied by "blistering," that is, the elevation of the tissues between the main veins and curling downward of the margins and tips of the leaflets. On susceptible blackcap varieties, the symptoms are more striking than on reds. Infected tips are stunted and curved, with a dark brown to black stripe along the concave side. Fruiting laterals are shortened, and the fruit is seedy and of poor flavor. In the second or third year, the new canes are short, reaching a height of only 1 or 2 feet; the internodes are shortened so that a rosetted condition is produced. Necrotic areas on the leaflets of blackcaps are also produced in some cases. Cooley has observed a decided retardation of foliation in green mottle mosaic plants. The diseased plants do not leaf out until the foliage on normal plants is well advanced.

On red varieties, the typical mottling of the leaves usually occurs, but the plants are not badly stunted and may continue to live for several years without great loss of vigor, although the yield and quality of the fruit are much reduced.

Transmission of the green mottle mosaic virus seems to be mainly by *Amphorophora rubi*. This aphid is a large, very active insect which is commonly found on raspberries, but is hard to detect because of its active habits as contrasted to the sluggish *Aphis rubiphila*. Repeated attempts by Bennett and others to transmit this mosaic virus by *Aphis rubicola* have all been negative. In 1932, Bennett reported the successful transmission of mosaic virus by another large aphis, *Amphorophora sensoriata,* and by *Amphorophora rubicola.* He was of the opinion, however, that *Amphorophora rubi* was the only insect of importance in transmission in nature.

Temperature has a marked effect on the development of symptoms in the case of green mosaic. The symptoms are most marked during the early part of the season when temperatures are comparatively low. The growth which occurs during midsummer may show no evidence of the disease. A lowering of the temperature toward fall will again bring out the characteristic symptoms on the renewed growth which occurs during cool periods.

Geographic Distribution. Because of the confusion which exists in regard to the limits of the various mosaic types, it is difficult to determine the geographic distribution of the green mottle mosaic. It is evident, however, that this type is found throughout the United States and Canada in both red and black varieties. It is probably distinct from the "mosaic" of England and the European continent.

Yellow Mosaic. This mosaic is acknowledged by most investigators to be distinct from the complex of the green mottle type. It was first described by Rankin and Hockey in 1922 as "mosaic" in red varieties.

Symptoms. On black raspberries, yellow mosaic is more destructive than is green mottle mosaic. The plants are severely dwarfed soon after infection, and the fruiting laterals are more upright than normal. The foliage is distinctly yellow and the blistered areas are not so marked as in green mottle mosaic. The leaflets are longer and narrower than normal. The distinct yellow blotching of the foliage during midsummer is perhaps the most characteristic symptom of this disease. While green mottle mosaic symptoms are decidedly masked by high temperatures, yellow mosaic will develop symptoms at a higher temperature, although masking may occur under extreme conditions. In other words, yellow mosaic has a higher temperature maximum for symptom expression than has green mottle mosaic.

In the second year after infection, the plants are much weakened, the fruit is seedy, and the symptoms are more marked. Plants with yellow mosaic usually die after the second or third year. On black raspberries the retardation of foliation, in contrast to green mottle mosaic, is not marked.

While yellow mosaic is most commonly found on blackcaps, it is also generally prevalent on red varieties where the symptoms are not so pronounced and likely to be confused with green mottle mosaic. A combination of the two mosaics may occur in the same plant, which leads to further confusion.

Yellow mosaic virus has been transmitted by *Amphorophora rubi* but not by *Aphis rubicola.* The fact that *Amphorophora rubi* will transmit both types of mosaic viruses has complicated the problem of separating these two diseases in transmission studies.

Bennett, by a series of ringing experiments, has demonstrated that curl and both types of mosaic viruses move only through the bark, probably in the food stream. He demonstrated also that the green mottle mosaic virus is dispersed more rapidly through the plant than is the yellows mosaic virus and that these two could be separated on this basis by proper technique. The downward rate of movement was found to be 0.2 in. per hour. It was also demonstrated that insects which had fed 2 hours on diseased plants would transmit yellows virus, but that in the case of green mottle mosaic virus a somewhat longer period was necessary, since no infection was obtained until after 12 hours of feeding. Viruliferous insects quickly lose their power of transmitting the viruses of mosaic when successively transferred to healthy plants.

Often the diagnosis of virus diseases in the field is rendered difficult because of a number of complicating factors. Plants attacked by insects such as leaf hoppers, aphids, and red spider or by diseases such as mildew and leaf spots may fail to show the characteristic symptoms or may show a condition which resembles closely some of the virus-disease symptoms. Frost injury and spray injury also complicate the problem. High temperatures also mask symptoms. A combination of viruses in the same plant has led to many mistakes, even on the part of experienced inspectors.

Control of Bramble Virus Diseases

Successful control of virus diseases depends to a large extent on cooperative efforts of the state, the nurseries, and the commercial fruitgrowers. In very large fruit-growing areas, the growers may cooperate to solve their virus disease problems by raising their own plants under rigidly controlled conditions.

Resistant Varieties. The development of highly resistant varieties suitable for the diverse commercial needs is probably the most promising line of approach to the ultimate solution of the problem of control. Some varieties escape infection because vectors of the virus do not feed on them. Varieties resistant to some of the virus diseases are now available, but the problem of adaptability of a variety is a local one.

Nursery Inspection. Rigid nursery inspection by well-trained men has been successful in greatly reducing the menace to the commercial growers of bramble fruit. State inspection service to be effective must be thorough and carried out by well-trained inspectors. Since the nurseries often buy their plants from commercial growers, it is necessary to have rigid regulations concerning the inspection of commercial plantings which are used for this purpose.

Roguing. The commercial grower who wishes to remain in the business must be on guard against the dissemination of virus diseases in his plantings. It is necessary in this case for the grower to become acquainted with the symptoms of the principal virus diseases and to inspect his fields at regular intervals. Plants showing any evidence of the disease should be carefully dug, removed from the field, and burned within a short time after removal. A still better method is to flame the plants in place by means of a blow torch so as to destroy any aphids present and then remove the plants as above. Roots should be carefully dug out so that new plants will not be produced from these. Early-spring diagnosis of green mottle mosaic in black raspberries is facilitated by the fact that the leafing out of infected plants is delayed for 2 or 3 weeks. Roguing at this time is advisable since it may be done before insect vectors become active.

Mother Plantings. Because of the difficulty of inspecting large fields, it has been feasible in some cases for growers to cooperate and establish their own nursery for supplying healthy plants. Starting with virus-free plants, these mother plantings are established at some distance from the commercial fields, and in a region where wild brambles are eradicated. The state is usually willing to furnish inspection service for such plantings, and they may be kept reasonably free from virus diseases over a period of years by rigid inspection and roguing all suspicious plants.

Spraying. Spraying for the control of insect vectors has not proved to be a practical method of control, although some benefit should be derived from such spray applications as are made for other diseases and insects.

Removal of Wild Brambles. Wild red and black raspberries not only become infected with the various virus diseases but they also harbor the insect vectors. For this reason all brambles in the neighborhood of the berry plantation should be destroyed.

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CHAPTER 9

DISEASES OF GRAPES

THE GRAPE

No other small fruit is so universally grown as the grape. Early history records the use of the "fruit of the vine" for wine making, and all early horticultural writings refer to this crop as one of the most desirable fruits for cultivation. The Old World, or European, grape, *Vitis vinifera*, is the one referred to in these early records. Grape seeds have been found in the oldest tombs of Egypt, and ancient writings indicate that wine making was practiced at least 6,000 years ago. The native home of the European grape is not known but was probably in the area around the Caspian and Black Seas.

Norse voyagers gave the name of Vinland to eastern North America because of the abundance of the native wild grapes in the region, and the early colonists of Virginia and elsewhere wrote of the abundance of grapevines clinging to the trees. The poor quality of native grapes in eastern North America led to many early attempts to introduce European grapes. This effort was unsuccessful not because of lack of a congenial climate but rather because of the fact that they were soon destroyed by fungus diseases and insect pests which found this species more desirable than the native species on which they had become established. In the southwestern section of the United States, where dry summer weather reduced the hazards of fungus injury and mild winters prevented cold injury, the European grapes found a more congenial home. The Spaniards established vineyards around their early missions in this region, and since that time the great grape industry of the West has depended almost exclusively on the Old World grape varieties.

The grapes of eastern North America are derived mostly from two native species, *Vitis rotundifolia* and *V. labrusca*. From the latter such universally grown varieties as Concord and Niagara were derived. The Muscadine varieties derived from *V. rotundifolia* are grown mainly in the southern sections. Recently hybridization of native and European grapes gives promise for the development of superior and disease-resistant varieties for growing under eastern conditions.

The world-wide growing of grapes makes it the most important fruit industry from an economic standpoint. California alone produces about 3 million tons of grapes annually on a half million acres. As large as this production is, it is only about one-tenth of the world production. Wine from the grape is an important source of national income for some of the European countries.

Grape diseases and insects have played a very important and interesting role in the historical development of plant pathology. The introduction of Bordeaux mixture as a fungicide, the realization of the importance of introduced pests into new areas, and the value of disease- and insect-resistant plants are based on the efforts to save the grape industry.

Grapes are not subject to many diseases, but the few important ones are extremely destructive when allowed to develop unchecked.

Black Rot of Grape

The only generally used common name for this disease is "black rot." The spot on the fruit occasionally has been called "bird's-eye." Both French and German authors adopted the English word for this disease.

History. Like the downy mildew, black rot is evidently of American origin. It was probably indigenous on the wild grapes and Virginia creeper, from which it became established in colonial times in the vineyards of the eastern United States. A destructive rot of the berries which was undoubtedly black rot is referred to by practically all the early horticultural writers who dealt with the grape. References may be found in which the crop is stated to have been a "complete failure" in certain wet years because of "rot" or "black rot."

Turner of Jacksonville, Illinois, gave an accurate description of black rot in several papers published in the *Horticulturist* between 1848 and 1852. This same writer in 1868 states, "A year or so after this time, about 1854, under the lens of a powerful solar microscope, at the Farmer's College near Cincinnati, Ohio, I examined grapes in various stages of blighting, which fully convinced me that in the case of grapes, the disease was wholly of fungoid, or cryptogamous origin." Engelman in 1861 also gave an accurate description of the disease, with a more detailed description of the fungus. It was not until after the introduction of the disease into Europe that any noteworthy work was done on it by American mycologists.

The importation of *Phylloxera*-resistant American rootstocks into France not only accounts for the introduction of downy mildew but also of black rot into Europe. The disease was first noted in France in 1885 but probably was present several years prior to this date. In 1887 Viala, a noted French viticulturist, visited the United States and with Scribner published in 1888 a bulletin on black rot.

Numerous publications by French, German, and Italian writers appeared after the introduction of the disease into Europe. These publications repeated much that had been published by Viala or dealt with experimental control methods following the experience with Bordeaux mixture in the control of downy mildew.

In 1911 Reddick published a comprehensive bulletin on the disease which was not only a summary of the past work but also a detailed account of the life history of the fungus and the results of experiments on control under New York conditions.

Geographic Distribution. The black rot organism is known to occur on a number of wild species of grape throughout the United States and Canada east of the Rocky Mountains. It is also prevalent on cultivated varieties throughout the same range. It does not occur in the dry grape-growing regions of California since humid conditions are necessary for its development.

After its introduction into France about 1880 it spread to all grape-growing sections of Europe and is now found in all countries where climatic conditions are favorable for its development although Trinchieri and others claim it is not found in Italy, from which country it has been excluded by rigid quarantine laws.

In general, it may be stated that black rot is more destructive in warm and humid regions than it is in the drier, cooler regions.

Economic Importance. Black rot is considered the most serious of grape diseases in all regions where it commonly occurs. Before the introduction of effective control measures it was not uncommon to have complete destruction of the crop in a season favorable for the disease. "Rot years" were fairly common, and the grower was helpless after the disease became established. Since it is most destructive in warm humid regions, the early vineyardists in the southeastern United States and along the Ohio and southern Mississippi Rivers soon discovered that grape growing was very hazardous. In dry climates the disease is never destructive, and in most of the grape-growing sections of the north and northeastern United States a number of years may elapse without serious loss.

Loss. The principal loss results from the direct destruction of the berries. This loss may occur early in the season through the blasting of the blossom clusters or when the berries are just formed, in which case the growers are not so aware of the cause of the failure of the fruit to set, or it may attack some of the berries when they are half- or two-thirds-grown. In this case the appearance of the bunch is often the most serious phase of the loss since the rotting of a number of berries on the bunch detracts from their appearance and consequently reduces their market value. Ordinarily loss of foliage is not severe, and the stem lesions, while they weaken the plant, rarely cause the death of the cane.

According to estimates of state pathologists the loss in southern states averages about 20 per cent of the crop, while in the northern states it rarely exceeds 10 per cent and usually varies from no loss to 8 per cent.

Symptoms. All the new growth of the vine is subject to attack during the growing season. On the *leaf* black rot appears in the late spring as a definite necrotic spot which at first is circular in outline and red in color. The lesions are not visible on the undersurface of those varieties which have a downy or hairy coat on this surface, but later the spots may be seen on either surface. The spots are usually on the thin part of the leaf blade between the veins and are usually scattered over the surface but may be grouped and numerous. Later, when the spot attains a diameter of about 1/16 in., the margin appears as a black line, while the remainder of the spot is brown. Another deeper-brown area may appear around the edge with a second black line beyond. The center remains a reddish brown to a grayish tan, depending upon the age of the necrotic tissue. The outline may vary from circular, owing to more rapid growth or to the presence of veins or veinlets.

On the upper surface of the spots appear a few minute black dots frequently arranged in a rather definite ring near the outer edge of the brown portion of the spot. These do not appear until after the lesion has attained some size and usually not until the outward growth of the fungus has ceased. The spots are usually about 1/8 to 1/4 in. in diameter, but on some varieties may be larger (Fig. 73). The black fruiting bodies are easily visible to the naked eye and, when the leaf is wet, are conspicuous.

Black rot lesions also occur on the *tendrils*, *leaf stems*, and *veins* of the leaf. These resemble those on the shoots described below. On the *shoots* the lesions are purple to black, somewhat depressed and elliptical to elongated. With the growth of the canes the bark may split along the long axis of the lesion. The dark fruiting bodies are evident in these lesions but are not so definitely arranged as on the leaves. Similar lesions occur on the flower stalks.

On the *berries* the lesions do not ordinarily occur until after the berries are half grown, but in some cases they are attacked earlier. The first appearance of the spot is a whitish area about 1/16 in. in diameter. This is soon surrounded by a ring of brown which widens rapidly. At this stage the dark ring about the lighter center gives a bird's-eye effect. The center of the spot becomes depressed with the softening of the tissue, so that when the diameter is 1/4 in. or somewhat larger, the lesion has a saucerlike appearance. About this time the black fruiting bodies may appear near the center of the lesion, although in some cases the berries may be almost completely rotted before these appear. The whole berry soon becomes involved, and shrinkage progresses rapidly, until finally a much-wrinkled mummy is produced, studded over with the numerous fruiting bodies. The whole berry becomes coal black as the rot progresses, thus giving rise to the common name of the disease. The diseased fruit "shells" easily, but it is not uncommon for the mummies to cling to the bunch throughout the winter.

Anthracnose and downy mildew on the berries may, in some cases, be confused with black rot in the early stages. A careful study of the symptoms will reveal decided differences by which these three diseases may be separated.

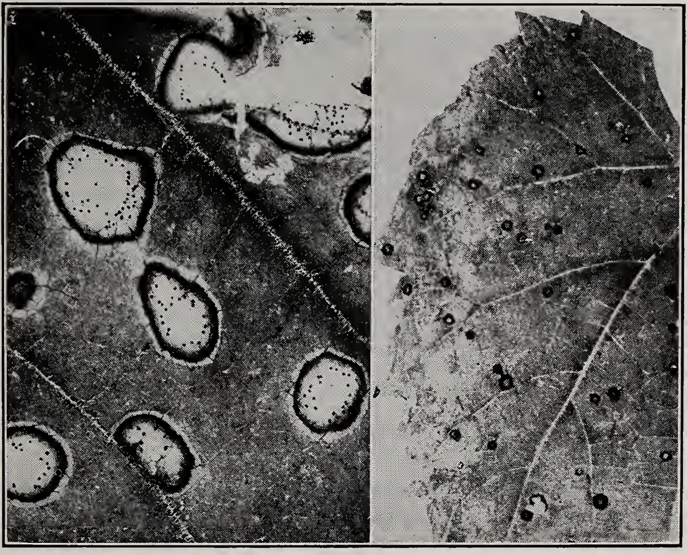

Fig. 73. Black rot of grape on leaf. At right, appearance on upper surface; at left enlarged to show pycnidia and dark outer border of lesions.

Causal Organism. *Nomenclature.* Owing to the fact that the leaf spot was not connected with the fruit rot by the early mycologists, a number of separate scientific names have been proposed for the two forms. The imperfect stage was described under a number of specific names of the genera *Phoma* and *Phyllosticta* (*Phoma uvicola* B. and C., 1873; *P. uvicola* var. *labruscae* von Thümen, 1878; *Phyllosticta labruscae* von Thümen, 1878; *P. ampelopsidis* Ellis and Mart.). In 1880 the perfect stage was described by Ellis from specimens furnished by Bidwell of New Jersey, and in his honor it was named *Sphaeria bidwellii.* This was transferred to the genus *Physalospora* by Saccardo in 1882. Viala and Ravaz in 1888, after a study of the perfect stage in France, transferred it to the genus *Laestadia,* but in 1892 these same authors assigned it to the genus *Guignardia* on the basis that the name *Laestadia* was preoccupied. Since the scientific name of an ascomycetous fungus is based solely upon the first description of the perfect stage, the name of the black rot organism becomes *Guignardia bidwellii* (Ellis), Viala and Ravaz.[1]

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[1] Class Ascomycetes, Order Sphaeriales, Family Mycosphaerellaceae.

Hosts. *Guignardia bidwellii* occurs on most species of *Vitis* (grape) and on *Parthenocissus quinquefolia* (Virginia creeper) and *P. tricuspidata* (Boston ivy). On the cultivated grapes it is most destructive on *Vitis vinifera* and its hybrids. The wild American grape species show no great resistance to the disease, and *V. labrusca,* from which many of the American grapes originated, is quite susceptible.

Morphology and Cultural Characters. *G. bidwellii* is an Ascomycete with three types of spores, ascospores, pycnospores, and spermatia. The *mycelium* is at first hyaline, much branched, with frequent septa. Old mycelium in the host tissue as well as in culture is brown under the microscope. In culture the whole growth is dark gray. As the fruiting bodies are formed, the mycelium in the neighborhood turns dark brown and finally black.

The *perithecia,* formed in the mummies, are supposed to be transformed pycnidia or pycnosclerotia, as described later. The shape of the perithecium is in general globose, but is somewhat irregular and uneven in outline. The ostiole is not prominent. The wall consists of an indefinite number of cell layers, often merging into a sclerotial-like growth, in which two or three perithecia are embedded or fused.

The *asci* are eight-spored, clavate, thick-walled, and measure 62 to 80 by 9 to 12 μ when examined in a dry mount. In water they elongate rapidly preparatory to the discharge of the ascospores. According to Reddick, no paraphyses are to be found in the perithecia, although they have been described by other writers.

The *ascospores* are hyaline, nonseptate, subovoid, or elliptical, slightly flattened on one side, 12 to 17 by 5 to 7 μ.

Pycnidia are produced in abundance on all organs with extreme rapidity. They are similar to the perithecia in structure, but the walls consist of only a few layers of cells, and the outline is more regular, especially in the pycnidia on the leaf. They are from 80 to 180 μ in diameter. *Pycnosclerotia* are formed from the pycnidia or from sclerotial masses of cells during the latter part of the season. These differ from the active pycnidia in that the walls are thicker and the contents consist of a number of large indefinitely defined cells. The pycnosclerotia later develop into perithecia or remain sterile. Similar structures of the apple blotch fungus produce pycnospores in the spring.

Pycnospores are borne on short sporophores and, under favorable conditions, so abundantly that large masses are forced out of the short ostiole, forming balls, or thickened, cylindrical tendrils. They are hyaline, globose to ovoid, continuous, 8 to 11 by 6 to 8 μ. The contents are granular, but contain a number of refractive globules (guttules). Several species of *Phyllosticta,* to which genus the imperfect stage of *G. bidwellii* belongs, show this and similar characteristics which set them apart from most of the common species of the genus. Among these may be mentioned *P. solitaria, Guignardia vaccinii,* and *G. aesculi.* In addition to the rather large guttulate spores, these also are characterized by a gelatinous appendage which occasionally may be observed.

Disease Cycle and Host Relations. In the spring, the perithecia are developing on the mummied berries which cling to the vines and those on the surface of the ground. Perithecia are not produced on buried mummies. The trimming of the vines by the vineyardist usually shakes off the clinging mummies, which are then in an excellent position to become soaked by spring rains as they lie exposed on the surface of the ground. Discharge of the ascospores occurs only when the mummies are thoroughly wet. The asci swell rapidly to almost twice their normal length, the ascospores move to the top of the ascus, where the terminal one presses against the inner wall, and by some internal force finally breaks through, the others following in rapid succession. By the force of this discharge, they are thrown some distance from the surface, where currents of air carry them to the developing plant parts. There is a continuous production of asci and ascospores throughout the entire spring and summer, thus providing for continuous primary infection, although the heaviest discharge is during the spring months. The ascospores alight on the growing parts and seem to be provided with an adhesive coat which sticks them firmly to the surface with which they make contact. The ascospores germinate slowly, often requiring 36 to 48 hours. Usually only a single germ tube is produced, and this generally arises from the side of the spore.

The method of penetration of the germ tube has not been determined, but it is reasonably certain that it is able to penetrate directly through the cuticle. Appressoria are known to be formed. After penetration, the germ tubes of both the ascospores and conidia ramify through the tissues of the host, but killing the host cells in advance of the penetration, thus indicating the production of a toxic substance. No haustoria are produced, and the mycelium is both intra- and intercellular, according to Reddick. The period of incubation varies with weather conditions, but in all cases is fairly long, indicating a slow advance of the mycelium after infection. Eight to twenty-five days are required for the first evidence of the lesions after infection.

The leaves are susceptible to infection only during the younger stages, when they are growing rapidly. Infection may occur as early as any growing parts are exposed. The primary infection usually occurs on the young leaves and on the fruit pedicels. Here there is a rapid production of pycnidia. The pycnidia are soon filled with pycnospores, and during rainy weather these issue from the ostiole in great quantities. Dissemination of the pycnospores is largely through meteoric water, since the spore mass is viscid but dissolves readily in water. Pycnospores are not exuded in the absence of abundant moisture.

Germination of the pycnospores is rather slow, requiring from 10 to 12 hours during favorable conditions. The pycnospores have an unusually long period of vitality. Several investigators have stated these spores will survive the winter and will germinate readily the following season. This is a possible additional source of primary infection in the spring, since the pycnidia on the cane lesions are in an advantageous position to bring about infection by the washing of the pycnospores on to the developing plant parts.

Secondary infection takes place throughout the season when weather conditions are favorable and when susceptible plant parts are present.

Fruit infection usually occurs after the berries are half-grown and before they have enlarged to their full size. It is generally held that the ripening berries are for some reason more resistant to infection.

About the first of August the production of pycnospores ceases and the pycnidia are transformed into pycnosclerotia, which in turn gives rise to the perithecia.

Relation to Environment. Black rot is a disease which develops best in a warm, moist climate. The fact that the mummies and lesions have to be soaked to permit spore discharge, together with the slow germination of the ascospores and pycnospores, indicates that infection is not likely to take place except when rather extended periods of rainy and cloudy or foggy weather prevail. This has been borne out by experiments in which meteorological data were accurately recorded. A period of rain, or of rain followed by foggy weather of 2 or 3 days' duration, is necessary for infection.

Little information is available as to the relation of temperature to spore germination. The fact that most infections occur during the late spring and early summer months would indicate a rather high optimum temperature for spore germination. Scribner and Viala mention the fact that spores held at a temperature of 25°C (77°F) germinate well, and in other tests they used temperatures of 30 to 35°C (86 to 95°F), which is relatively high for optimum spore germination.

Control. *Sanitation and Cultural Practices.* Grapevines are ordinarily pruned severely each year during the dormant period. At this time practically all mummies which might have clung to the vine are removed or shelled off. In addition, many of the canes produced the previous season on which lesions may be present are cut off and removed from the vineyard. This practice undoubtedly aids greatly in the control of black rot, but the sheltered berries are advantageously placed for the production and discharge of the ascospores. It has been found that mummies covered with earth do not produce ascospores, or if they are produced they would not reach the developing vines. For this reason, cultivation of the vineyard early in the spring is advocated when practical. If, in the process of cultivation, the earth can be thrown in under the vines, thus more effectively covering the berries, better control may be expected.

Since the spores require a moist surface over a rather long period for germination, any practice which allows free circulation of air and rapid drying of foliage reduces the chances of infection. The canes, therefore, should be kept well above the ground, and weeds should be kept cut beneath the vines.

Varietal Relationship. Attempts to grow European (vinifera) grapes in the southern part of the United States have failed, owing to their susceptibility to black rot and downy mildew. American grapes which are hybrids of vinifera are likely to be very susceptible to black rot. Grapes of pure American origin vary somewhat in their susceptibility. Certain commercial varieties have been discarded in the past by practical vineyardists because of their lack of resistance to black rot. Varieties with large juicy berries are most susceptible, while those ripening late in the season are, in general, the least susceptible. The commercial varieties now generally grown are sufficiently resistant when protected during rainy periods with effective fungicides.

Spraying. The fortunate discovery of Bordeaux mixture in France for the control of downy mildew of grape and its general use in that country at the time of the introduction of black rot demonstrated rather quickly that it could be used successfully for the control of the latter disease. The early experiments in France were not entirely successful, because of the fact that the life history of the fungus was not thoroughly understood. The early experiments by the U.S. Department of Agriculture were also directed against downy mildew, but as in France, the effect on black rot was also noticed, and the control of this disease was soon included in the experiments. After trying a large number of copper sprays, Bordeaux mixture was found to be the best for general use on the vine. With the introduction of lime sulfur some attempts were made to use this material, but it was soon found that it would cause serious injury under certain conditions, especially where high temperatures prevailed.

A detailed study of the life history of the organism by Reddick and others demonstrated that spraying must be timed in such a way as to anticipate a rainy period. Since the black rot fungus is capable of infecting throughout the growing season or until the berries reach full size, the cost of spraying would be excessive if the vines were kept covered throughout the season. Another important consideration is the necessity of spraying early in the season to protect the young shoots as they develop. In a dry season, after the application of the first spray, i.e., when the young shoots are 12 to 18 in. long (a week before the bloom), money can be saved by timing subsequent sprays according to the weather. Where certain insecticidal sprays are necessary, the introduction of fungicide regardless of weather conditions may save labor.

Ferbam has given very good control of black rot and has replaced Bordeaux mixture in most spray schedules.

Bagging the fruit clusters with cheap paper bags successfully protects them against black rot infection. Where a few vines are grown, this practice will save the cost and trouble of spraying.

Black rot is not an important disease in transportation and storage, since it is not likely to develop on ripe grape.

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Downy Mildew of Grape

Two diseases of the grape are commonly called mildew. "Surface," or "powdery" mildew is not nearly as destructive in most regions as is the "downy" mildew here described. In early horticultural literature these two mildews were sometimes confused. In a few early accounts the name "brown rot" was used for the fruit infection, but this name has been used for anthracnose, an entirely different disease, and consequently is not descriptive. In France downy mildew is called "le mildiou" and in Germany "falscher Meltau."

History. Downy mildew is undoubtedly of American origin and was probably indigenous on the wild grapes of the eastern United States, from which it spread to the cultivated vineyards when these were established by the colonists. It was not known to occur outside America until after 1870, although it was first described from this country as early as 1834. In the early part of the last century we find various horticultural writers referring to the destructive action of the mildew on cultivated grapes. Its history in Europe dates back to the time when French vineyards were suffering from the grape *Phylloxera,* an insect pest also introduced into Europe from America. It was found that rootstocks of the American species were more resistant to this aphid than was the European vinifera grape, and consequently about 1870 importations of the American vines were made into Europe, and somewhere around 1875 the downy mildew fungus evidently was introduced and became established, for by 1878 it was prevalent enough to cause alarm. Since the European vinifera grapes were far more susceptible to this disease than were the American species, it soon became a real menace to grape culture throughout the humid sections of Europe.

The serious threat to the grape industry in Europe gave rise to a series of investigations by French research workers. The contributions of Cornu (1882), Millardet (1883), Cuboni (1887), and Viala (1893) cleared up many details in the life history of the organism, but probably the most significant result was the accidental discovery of the value of a mixture of lime and copper salts in the control of the mildew. Millardet, in studying the disease, noticed that many vines along the roadside at Medoc had retained their foliage, while those farther from the road were completely defoliated. On inquiry he found that the growers were in the habit of sprinkling these vines with a mixture of verdigris (copper acetate) and lime or with copper sulfate and lime in order to make the grapes unattractive to passers-by who were in the habit of picking the fruit. Following up this clue he tried various combinations of copper salts and lime and thus introduced Bordeaux mixture.

In America Farlow, in 1876, gave an excellent botanical description of the fungus, including a detailed study of the germination of the conidia and the formation of swarm spores. Between 1886 and 1889 Scribner of the U.S. Department of Agriculture issued a series of reports on mildew and black rot. These reports not only recorded numerous experiments in various parts of the country on the control of the disease, but also contained translations of a number of French and Italian articles and many points of historic interest. He followed very closely the results of the French trials of copper compounds, and it is significant that within 5 years after the discovery of Bordeaux mixture in France, many grapegrowers in America were using this material for the control of both downy mildew and black rot. One statement in these reports is especially significant as bearing on the history of the disease. Howell of South Carolina wrote: "What is known as the downy mildew of the vine (*Peronospora viticola*) which causes 'brown rot' has been known to this state from time immemorial."

Gregory of Cornell University in 1915 published the results of his extensive experiments in which he determined the method of germination of the oöspores and cleared up many important points in the life history and pathogenicity of the organism. Further information in the history of this disease will be found in subsequent sections.

Geographic Distribution. Downy mildew is now found in most regions of the world where grapes are grown under humid weather conditions. Originating in America, it was introduced into France during the seventies and soon spread over the Continent and to northern Africa. It was reported from South Africa in 1907, from Australia in 1917, and from New Zealand in 1926. It is also known to occur in several countries in South America and in Japan, China, India, Asia Minor, and Russia.

In the United States it occurs in all regions east of the Rocky Mountains, but is most destructive in the humid regions in the northern section of the Mississippi Valley, along the Atlantic seaboard, and about the Great Lakes. It has never become established in the great California grape-growing areas, in spite of the fact that the vinifera grape which is especially susceptible is extensively grown. This is probably due to the dry conditions, under which the fungus does not thrive.

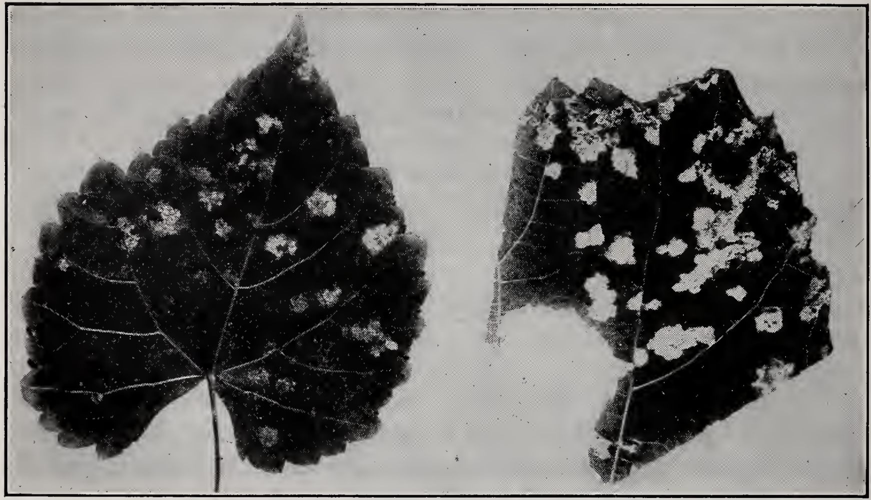

Fig. 74. Downy mildew of grape. Left, upper surface showing dead tissue opposite the downy spots on the lower surface (right).

Economic Importance. Loss from downy mildew is more likely to result from the effect on the vine than from direct rotting of the fruit, although the grower is not likely to realize this fact fully. If the disease occurs early in the season, the young bunches of berries may be entirely killed by the attack of the stems as well as of the young fruit. If the disease appears later in the season, the berries are not so likely to become infected and the main loss comes through defoliation and killing of leaf tissues, which results in the failure of the fruit to ripen properly if at all. Fruit from vines seriously diseased has a low sugar content. Another serious loss results from the attack of young shoots, which are consequently dwarfed and do not make the canes necessary for bearing the crop. Under very favorable conditions half-grown or older berries may be infected, resulting in the production of unsightly bunches. In France, before the control of the disease was worked out, it was not uncommon for entire vineyards to be so completely defoliated as to result in a total loss of the crop. Ordinarily the loss from downy mildew is not serious in the regions where rainfall is light and humidity is low. The loss over a period of years is not likely to be great since epidemics occur only during those summers when rainfall is unusually heavy at critical periods. From 50 to 75 per cent of the crop may be destroyed where no protection is given, provided weather conditions are favorable.

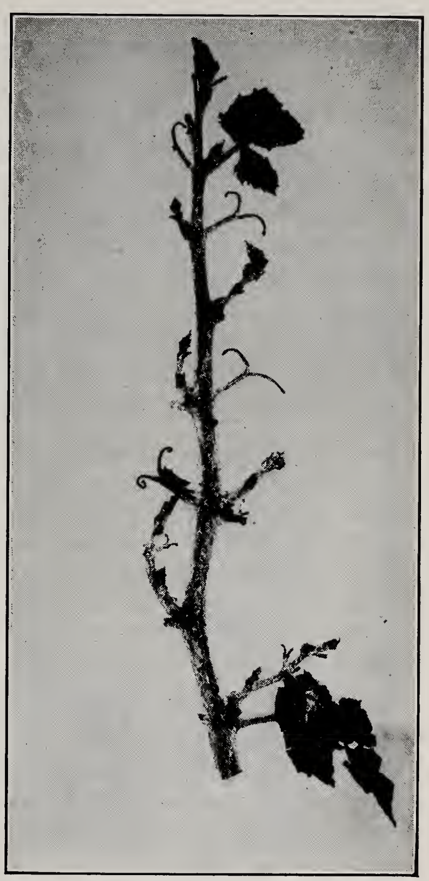

Fig. 75. Downy mildew of grape on shoot and tendrils.

Symptoms. The disease may appear on any succulent part of the vine. It is commonly found on the leaves, young shoots, and immature fruit clusters. On the *leaf* it first appears as a pale yellow, rather indefinite spot on the upper surface. The translucent character of the young stages of the spot has given rise to the name "oil spots." If the variety attacked has a hairy undersurface it is difficult to detect the affected area on this surface, but on some wild species having few hairs the white aerial growth of the fungus can be seen as soon as, or before, the yellow area is evident on the upper surface. The downy appearance of the spots gave rise to the common name (Fig. 74). Later the leaf tissue is gradually killed, and the necrotic lesions become light to dark brown and irregular in outline. The white color of the fruiting mass on the undersurface turns to a dirty gray. Frequently, when the spots are numerous or the fungus especially vigorous, the dead areas merge into each other so that a large section of the leaf between the main veins may die and turn brown.

Young *shoots* attacked by the fungus are decidedly shortened but are thicker than normal shoots owing to hypertrophy. The whole shoot, including tendril and young leaves, often becomes covered with the white, fruiting structure of the fungus (Fig. 75). After a period of growth in which the whole shoot may become decidedly distorted, the tissues turn brown and die, preceded by the collapse and disappearance of the downy growth on the surface. If the attack is localized on a portion of the shoot or the variety is somewhat resistant, the cane may survive, but it frequently shows the effect of the attack in the distorted appearance of the growth.

Flowers and young fruit clusters may be attacked, in which case usually the entire cluster is invaded, and after the usual appearance of the downy fruiting masses, the entire cluster dies. In some cases only a portion of the cluster is attacked and the remainder may remain alive and develop. If infection occurs on the fruit when young, it is usually covered with the downy growth, but when the attack occurs after the berries are half-grown, the rotting takes on an entirely different character. In the latter case the growth of the fungus is largely internal, the berry turns to a dull green, then to a brown, and finally the berry wrinkles somewhat but does not take on the deeply shrunken appearance and black color of fruit attacked by the black rot fungus. This type of fruit rot is often called "brown rot." Berries attacked by the downy mildew are inclined to "shell" and are rarely mummified on the vine.

Causal Organism.[1] According to Wilson (1907, p. 408), the first collection of the fungus was made by Schweinitz in America in 1834. In 1848 it was named *Botrytis viticola* by Berkeley and Curtis. De Bary in 1863 redescribed it as *Peronospora viticola.* In 1886 *Peronospora* was separated into two genera by Schroeter, and the grape mildew organism was referred to the genus *Plasmopara* by Berlese and De Toni in 1888. Wilson in 1907 subdivided the genus *Plasmopara* into *Rhysotheca* and *Plasmopara,* but this division has not been generally accepted. If accepted the name of the downy mildew would be *Rhysotheca viticola.*

Hosts. *Plasmopara viticola* attacks all common species of wild and cultivated grapes (*Vitis* spp.). In addition it has been reported on the Virginia creeper (*Parthenocissus quinquefolia*) and on Boston ivy (*Parthenocissus tricuspidata*). The European grape (*Vitus vinifera*) is more susceptible than the cultivated American grapes which are derived from native species. In some hybrids of the American and European species the lack of resistance to downy mildew is evident.

Morphology. The *mycelium* is nonseptate, except occasionally in the older hyphae. In the host tissue, where it is intercellular, the hyphae are very irregular, both as to diameter and branching. Often the threads conform to the general outline of the spaces in the cellular structure traversed by the fungus. For this reason the diameter of the hyphae may vary from 1 to 2 μ to as much as 50 to 60 μ within a short space. The mycelial wall is thin, the contents hyaline and granular. Older portions of the hyphal growth may be entirely empty of contents, while the advancing young threads are densely filled with the granular protoplasm.

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[1] *Plasmopara viticola* (B. & C.) Berl. and DeT., Class Phycomycetes, Order Peronosporales, Family Peronosporaceae.

Haustoria are produced in abundance (Fig. 76*B*). These enter the cell through a very small opening in the cell wall made by a small tube arising from the mycelium growing alongside the cell. Inside the cell wall this tube expands into a globose structure which presses back the plasma membrane but does not pierce it. Where the haustorial tube enters the cell, the cell wall is decidedly thickened and a collar, composed of remnants of the invaginated cell wall, may be seen around the base of the swollen haustorium.

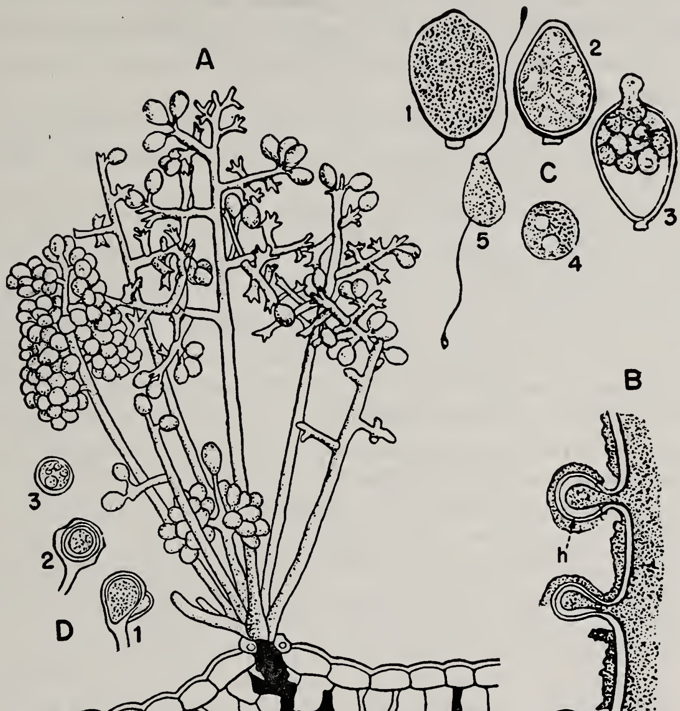

Fig. 76. *Plasmopara viticola. A*, group of conidiophores arising from stomatal opening on undersurface of leaf, showing conidia on sterigmata of branched conidiophores; *B*, haustoria invading host cells; *C*, conidia showing formation of zoospores and single zoospore with flagella (5); *D*, 1, oögonia with antheridium, 2 and 3, oöspores.

A cushion of swollen mycelial segments is formed beneath the stomata preparatory to the production of the *conidiophores*. From these swollen bodies arise many smaller ones which push upward through the stomata. From 1 to 20 conidiophores may arise through a single stomatal opening (Fig. 76*A*). The base of the conidiophore is constricted where it passes between the guard cells of the stoma, but swells again at the surface. In some cases the conidiophores may be pushed directly through the epidermis, and in the young fruit they arise from lenticels. Conidiophores are produced most readily in subdued light or at night under humid conditions. On the leaf, with rare exceptions, they are formed on the undersurface only. This is due in part to the absence or rare occurrence of stomata on the upper surface.

The conidiophores are from 300 to 500 μ long and 7 to 9 μ in diameter when measured above the swollen base. The branching of the conidiophores is characteristic of the species. From four to six monopodial branches arise at rather definite intervals along the upper portion of the conidiophore, the final branch being formed by the terminal portion. These primary branches in turn form two to three secondary branches. The lower branches may in turn form short tertiary branches. From the final branch two or three short, blunt sterigmata arise on which the conidia are borne.

The *conidium* is formed by the swelling of the end of the sterigma, into which the protoplasm from the conidiophore flows until the spore attains about normal size, when it is cut off by a septum.

The hyaline, thin-walled conidium is usually ovoid with a small papilla at the tip which gives it a lemon-shaped outline. The conidia are 11 to 18 μ by 15 to 30 μ in size, but variations both in shape and size are common (Fig. 76*C*).

Germination commonly occurs by the formation of *zoospores* inside the conidium (Fig. 76*C*). Two other types have been described, one the formation of a germ tube and the other the emission of the entire contents of the conidium, followed by the formation of zoospores from the extruded mass of protoplasm. When the zoospores are formed within the conidium the protoplasm separates into indefinite masses. These masses then round out into definite zoospores which separate and move about within the conidial chamber. The swarm spores escape through an opening in the tip of the conidium. The zoospores are pear-shaped with a depression on one side where the two cilia or flagella arise. They measure about 7 by 9 μ, and the flagella are about 30 μ long. After swimming about for a few minutes to a half hour, the zoospores come to rest and round off, and the flagella disappear. From this quiescent spore a germ tube arises to infect the plant.

Oöspores are formed in the host tissue (Fig. 76D). Commonly they are produced in the palisade cells or spongy parenchyma of the leaf very near to the principal veins. They have also been found in berries and in the cortex of the shoots. Oöspores are not formed abundantly, as in some other species of the downy mildews. The oöspores are from 25 to 36 μ in diameter surrounded by the thickened, persistent rough oögonial wall. Farlow pictures the *antheridium* as a long slender thread which partially encircles the oögonium. Other investigators have described a clavate structure which appears to be an antheridium, but Gregory states that if an antheridium is present in this species it is certainly rarely found, and in no instance has the fertilization of the oögonium or the formation of a definite impregnating tube been observed, as can usually be seen in other species of this genus.

Gregory has observed the germination of the oöspore. A single germ tube is produced which functions as a conidiophore. A single conidium is formed on the end of this, and in this sporangium swarm spores are formed and escape as described above. Arens confirmed Gregory's observations, but states that some oöspores have been found to require exposure through two winters before they will germinate.

Disease Cycle. *Plasmopara viticola* is an obligative parasite, that is, it develops only in the living host tissue. So far as known it does not live over winter in the live dormant parts of the plant. The only known method of overwintering is by means of the oöspores, although in Brazil where the grape is evergreen the fungus may continue to produce conidia throughout the year. The oöspores are embedded in the dead leaves which disintegrate during the winter, thus freeing the oöspores. These must be subjected to low temperatures before they germinate. The production of conidia and swarm spores from the germinating oöspores probably occurs during rainy periods when drops of water carrying them are splashed on the leaves near the ground. Primary infection having been established, conidia are abundantly produced on the lesions, and the disease may then spread throughout the vineyard. The conidia are easily detached from the sterigmata, and on disturbing the leaves, clouds of these are seen to be carried by currents of air to adjoining tissues. They may also be carried by water or mechanical means. The conidia germinate quickly, producing myriads of swarm spores, so that reinfection takes place very rapidly under favorable weather conditions. The swarm spore produces a germ tube which grows along the surface of the leaf until a stomate is encountered, when it enters the substomatal chamber through the opening. So far as known, infection takes place only through the stomata, and since these are on the undersurface of the leaf, all infections occur on this surface. After entering the substomatal chamber the germ tube swells and sends out branches, invading the surrounding tissue for some distance.

According to Gregory the incubation period of the fungus is from 7 to 12 days on susceptible varieties, but may be as much as 20 days on other varieties.

At the end of the season oöspores are formed in the old leaves, thus completing the life cycle.

Relation to Environment. Downy mildew is of economic importance only in localities where the rainfall is fairly high during the summer months and where humid conditions prevail. Since infection is dependent upon the presence of water both for germination and for dissemination of the zoospores, it is evident that free water must be present on the leaves for a considerable period of time. However, heavy dews may be sufficient to form the necessary water film after the dissemination of the conidia.

There is some difference of opinion as to the range of favorable temperatures for conidial germination. The optimum according to Gregory is between 50 and 60°F and a minimum about 40°F. At optimum temperatures germination of conidia may occur in ½ hour.

Conidia will germinate best in diffuse light or darkness, but contrary to the opinion of early workers, germination occurs in direct sunlight provided a low temperature is maintained. Conidiophores are almost invariably formed at night or in very subdued light.

Pathological Histology. Immediate death of the cells attacked does not occur in the case of downy mildew. This is characteristic of most of the obligative parasites and indicates that no toxin is produced. After the formation of the haustoria, the cell contents gradually change, with the loss of chlorophyll and plastids being the outstanding characteristics of parasitism. Finally, the cells become plasmolyzed and turn brown, and the characteristic necrotic area is produced.

The cortex, collenchyma, epidermis, medullary rays, and pith of the stems are affected, according to Gregory. The swelling of the stem mentioned above is due to the hypertrophy of the cortical cells and the presence of the abundant mycelium.

Control. Since the only known method of primary infection is by means of the oöspores which are in the old leaves, sanitary measures are of great importance in the control of this disease. Removal or plowing under of the leaves and keeping the canes as high above the ground as is practical serve to reduce the chances of spores being splashed on the new growth in the spring.

Free circulation of air by proper spacing and pruning, thus facilitating rapid drying, is also of importance in reducing the chances of secondary infection.

In regions where downy mildew is prevalent, the selection of varieties resistant to this disease is advisable, but in general the loss is not sufficient to warrant sacrificing desirable market qualities in order to attain this end.

Spraying. In regions where both downy mildew and black rot occur, the mildew is likely to be controlled by the sprays applied for black rot. Bordeaux mixture or other copper sprays are most effective in the control of downy mildew and should be applied under conditions where the disease is likely to cause serious loss. The time and number of applications depend upon local conditions. The first application should be designed to protect the new foliage from primary infection.

Where ferbam is being applied for black rot control it may be substituted for Bordeaux mixture, although it is not as effective for downy mildew.

Captan, 0.5 per cent, is reported (Switzerland) to be even more effective than Bordeaux in the control of downy mildew.

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Powdery Mildew of Grape

The term "powdery mildew" is generally used in English-speaking countries for this disease since this avoids confusion with "downy mildew," which is caused by an entirely different fungus. "Surface mildew" is a general term applied to diseases on many hosts caused by this group of fungi. On the European continent powdery mildew of the vine is called "oidium," from the generic name of the imperfect stage of this and similar surface mildews.

History and Geographic Distribution. For many years powdery mildew of grapes was supposed to be of American origin, but there is some evidence that it was introduced from Japan. It seemed not to have attracted much attention in Europe until after 1845, when it was first reported as occurring in England. It was found in France in 1847 and by 1851 was widespread in European countries. It is possible the disease was present much earlier but that it had escaped attention until it appeared in epidemic form during this mid-century period. It was known to be present in northern California on vinifera grapes as early as 1859. In 1861 a California grower successfully controlled mildew by the use of sulfur. Pierce in 1892 speaks of the "rapid spread" of the disease in the 1860s to the southern California vineyards.

Powdery mildew is now known to occur in all countries where grapes are grown. It is more destructive to the vinifera grapes than to the American species and is more of a problem than downy mildew in regions where relatively dry conditions prevail during the growing season.

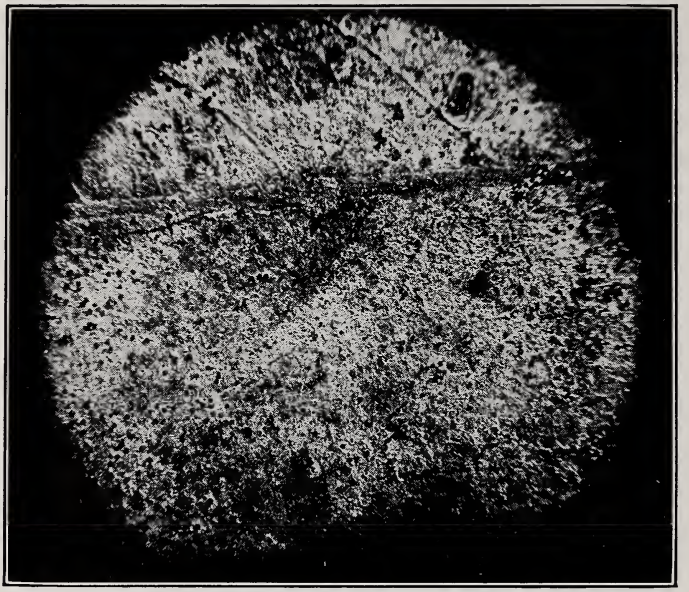

Fig. 77. Grape powdery mildew. Enlarged to show perithecia in webby growth of hyphae on leaf surface.

Symptoms. The disease may be found on any new growth but is most conspicuous on the upper surface of the leaves. Here small, rather indistinct, white patches appear which later have a powdery appearance. Usually these enlarge so that the entire upper surface of the leaf has a dusty coating (Fig. 77). In some cases the patches remain limited throughout the greater part of the season. Severely attacked leaves have a tendency to curl upward during hot, dry weather. On the cane the spots are more likely to be limited but have the same general appearance as on the leaves. Later in the season these areas become discolored, at first brown and later almost black. If the blossom clusters are attacked, the flowers may wither and drop without setting fruit, but such early infection is unusual. The young berries may show evidence of the disease either by spotting, as on the leaves, or by a growth of the fungus from the fruit stem over the tender portion of the berries or over its entire surface. The berries are often misshapen or show rusty spots which have corked over. Sometimes the fruit splits as a result of the retarded growth over the infected area. As the fruit approaches maturity it becomes immune to attack. While the fungus may develop on the undersides of the leaves, it is likely to be inconspicuous, especially where this surface is at all hairy.

Late in the season there may be numerous black specks on the surface of the infected spots, but in many regions these are not present. These are the sexual fruiting bodies, the perithecia, and are so small that they may be overlooked unless one uses a hand lens (Fig. 77).

Leaves attacked by surface mildew usually retain about their normal green color but may show some discoloration toward the end of the season.

Economic Importance. On American species powdery mildew rarely causes extensive damage. This is partly because it is late in the season before the disease becomes generally prevalent, and by this time the canes have made their full growth and the berries rarely show evidence of the disease. Furthermore, surface mildew, while present every year, rarely assumes epidemic proportions. In certain regions in New York when conditions are favorable, a loss of 50 per cent of the crop has been reported, mainly as a result of shelling of the fruit from early attacks of the fungus.

The damage to *vinifera* grapes may be very extensive. This is especially true in Europe and in western United States. In these regions powdery mildew is considered of greater importance than downy mildew. Shortly after its appearance in Europe in the fifties of the past century reports of almost complete loss of crops were frequent. The amount of space given to discussions of this disease in the literature since that time attests to its continued importance.

Causal Organism. Powdery mildew is caused by an Ascomycete, *Uncinula necator* (Schw.) Burr.[1] The imperfect stage was named *Oidium tuckeri* by Berkeley from specimens collected in England about 1845. Since the perfect stage is rarely found in Europe, the fungus and the disease were usually referred to as "oidium."

The fungus *mycelium* is entirely superficial and derives its nourishment from *haustoria* which penetrate the cuticle and epidermal cells. From the webby growth of the mycelium numerous short club-shaped conidiophores arise, from the apex of which a chain of *conidia* are cut off. These numerous beadlike chains produce the white powdery effect. The conidia are hyaline, ovate to oblong, nonseptate, and measure about 15 by 30 μ.

The *perithecia* are found late in the season on the leaves or stems. They may be scattered but are often thickly clustered on one area. Like other powdery mildews the perithecium is a closed structure; that is, there is no ostiole. Arising from the wall cells of the perithecium are a number (20 to 30) of long, narrow appendages with curled tips characteristic of the genus *Uncinula* (Fig. 78). Within the brown to black perithecium four to eight ovate asci are borne. These contain four to six hyaline, oval, nonseptate ascospores measuring 18 to 25 by 10 to 12 μ.

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[1] Class Ascomycetes, Order Perisporales, Family Erysiphaceae.

Hosts. *Uncinula necator* is confined to species of *Vitis* and *Ampelopsis.* It is of economic importance on grapes of which *V. vinifera* seems to be most susceptible.

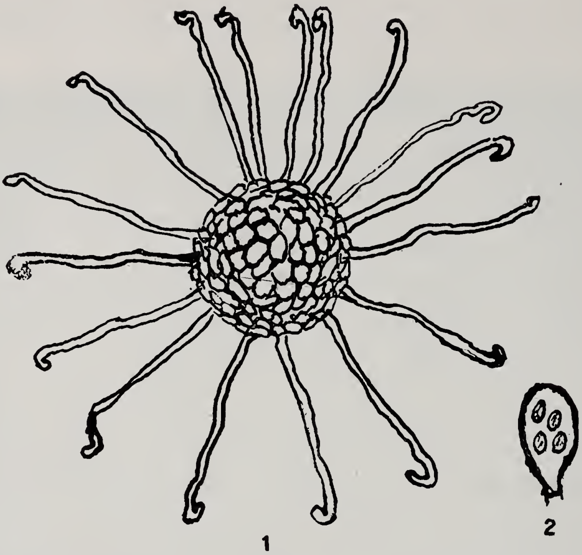

FIG. 78. Perithecium and ascus of *Uncinula necator* showing at 1, hooked appendages; at 2, ascus with four ascospores.

Disease Cycle. The method of overwintering of the fungus has not been satisfactorily determined. Some are of the opinion that spring infection is mainly from the ascospores released from the perithecia, while others, basing their argument on the frequent absence of the perithecia, claim that conidia survive the winter or that the mycelium remains alive in sheltered places on the shoots. The ascospores in the closed perithecium are released in the spring by the swelling and rupturing of the perithecial wall. The perithecia may be on the fallen leaves or on the infected shoot. Primary infection results when these spores alight on any green surface of the developing vine, and conidia are soon formed over the infected area. The powdery spores are easily wafted to neighboring leaves or other organs to bring about secondary infection. The fungus mycelium may spread over large areas of the leaf from a single point of infection.

If conidia survive the winter the same sequence of events occurs.

Haustoria are responsible for a certain amount of browning of groups of epidermal cells through the withdrawal of nourishment and water, but general death of the leaf tissue does not occur. Hot weather may cause a certain amount of curling and scorching of the infected leaf.

Unlike downy mildew, powdery mildew is considered a "dry weather" fungus and is often present in dry areas or in dry seasons when downy mildew is absent. Temperature also influences its growth. Between 70 to 90°F the fungus grows rapidly but is slowed down above and below these temperatures. The mildewed leaves usually appear first on shaded portions of the vines.

Control. Cultural practices are important in the control of powdery mildew. Rapid drying of vines following dews and rains reduces the chance of infection. For this reason an effort should be made to train the developing shoots in such a way as to reduce shading and allow air circulation.

"Sulfuring" has been the standard practice for the control of this mildew in Europe and California since the middle of the past century. The superficial nature of the fungus permits actual killing of the mycelium even after it has started development. While sulfur either as a spray or dust can be successfully used on vinifera grapes in regions not subject to high summer temperatures, it is very toxic to American grape varieties when high temperatures prevail as in eastern United States. In these regions sprays or dusts used for the control of other diseases usually suffice to check powdery mildew. The time of application and material to use may vary widely in different regions, and the grower is advised to follow directions of experiment stations or experienced local growers. In New York, for example, Bordeaux mixture and fixed coppers are recommended.

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Grape Anthracnose

Anthracnose, also called "bird's-eye rot" from the characteristic lesions on the fruit, is regarded as the fourth most important disease of the grape in the United States.

History and Geographic Distribution. There is evidence that this disease was referred to by Pliny as occurring in Italy in the first century of the Christian Era. It was found on specimens in France collected in 1839. In America it was first reported by Burrill in Illinois, who stated that the disease was causing damage to grapes in central Illinois, and further adds: "But it certainly devastated our vineyards before the date mentioned and is now known to occur over wide areas of our country." He had observed it in Indiana, Michigan, and Ohio. It would seem that the disease, present for centuries in Europe, was introduced into the United States when the European grape was brought into eastern United States, since the fungus could easily be transported on cuttings or young vines. Thus America exchanged downy mildew and black rot, native to North America, for anthracnose, which was hardly a fair bargain for Europe.

Since 1900 anthracnose has been studied by a number of investigators both in America and Europe. The most elaborate of these studies was by Viala and Pacottet in France, published between 1904 and 1906, in which they reported among other details a number of types of spores and fruiting bodies in their cultures, indicating the polymorphic nature of the fungus.

In 1929 Shear announced the discovery of the perfect stage and placed the fungus in the genus *Elsinoë,* since it was very similar to the perfect stage of the raspberry anthracnose discovered by Burkholder and placed in this genus by Jenkins.

Aside from records of distribution and control measures, few additional contributions have been made to the earlier studies. The disease has now been reported from all regions of the world where grapes are grown. It is of importance only in those regions of relatively heavy rainfall during the spring months, and in most years often causes little injury. Fortunately, the Concord variety, extensively grown in North America, is quite resistant and rarely suffers damage.

Symptoms. The fungus attacks all parts of the plant in the green stage but is most often observed on the new shoots and fruit. On the *leaves* the first evidence is the appearance of small, irregular, dark brown spots. These later turn gray in the center with a border of dark brown tissue. The central tissue often falls out later in the season. Lesions similar to those on the shoot as described below often are found on the main veins and petioles of the leaves.

The most conspicuous symptoms are those on the *shoots* and *tendrils.* Here appear small isolated light brown spots, at first circular in outline but later elongating into elliptical, slightly sunken areas. The tissue at the border of these lesions becomes slightly raised, forming a darker-colored rim, while the central area turns to ashy gray. These lesions may be so numerous as to give a rough, scarred appearance to the shoot. Sometimes in midsummer during wet weather small pink spots may be observed in the depressed center of the lesions. These are spore masses of the causative fungus. The disease on the shoots, when severe, restricts its growth and in some cases causes dwarfing of the leaves, which become lighter green than the surrounding healthy leaves. Leaf veins and petiole infection may cause downward curling of the leaf, but rarely defoliation.

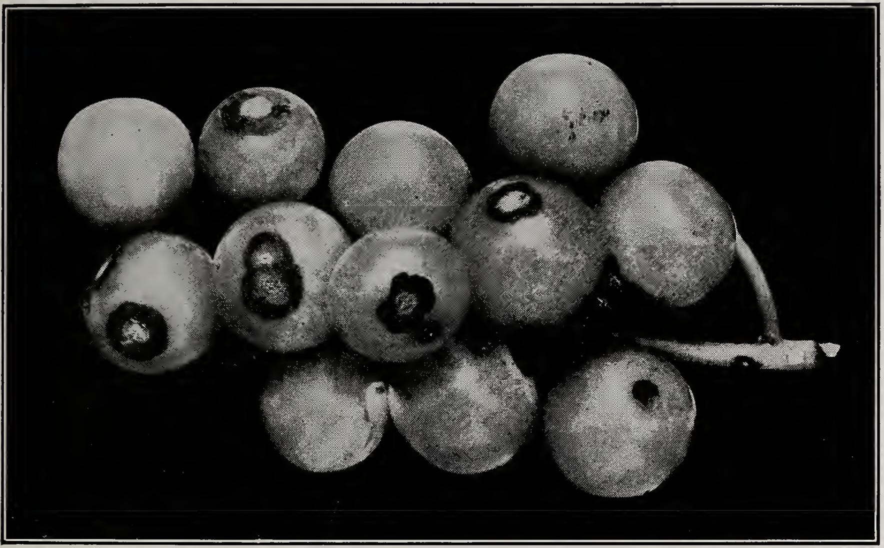

FIG. 79. Grape anthracnose on berries and fruit stem. (*Photograph by M. W. Gardner.*)

The disease on the *berries* is referred to as "bird's-eye spot" because of the gray center surrounded by a reddish-brown zone (Fig. 79). They start as small dark red spots which may appear on any part of the berry, but often only two or three well-separated spots are present. Unlike black rot there is only a very slight depression and no later shrinkage of the berry. The individual spot may enlarge to involve as much as half the berry but is often restricted to a lesion about ¼ in. in diameter. The flesh of the berry remains firm, and there is little evidence of softening of the interior tissues. However, the cells below the lesions become dry and hard, and further enlargement of the neighboring tissue results in a misshapen fruit. At times the fruit cracks, exposing the seeds. Scabby areas may appear on the mature fruit when the fungus is checked in an early stage. Fruit stems may have lesions similar to those on the petioles and tendrils. The old lesions are found on the 2- and 3-year growth when the vines are unpruned, but usually most of the previous year's wood showing the disease is cut out in pruning. The old lesions are on the stubs of pruned canes and are not very conspicuous, since these are often covered by an overgrowth of rough bark.

Economic Importance. Anthracnose appears sporadically; often when it is present on the shoots, fruit infection is absent. In regions where the disease occurs in a severe form, it may cause extensive damage to both fruit and vine on susceptible varieties. The growth of the shoots is restricted, resulting in poor-quality fruit. The fruit spot when severe may reduce the yield 50 per cent or more. Sometimes a few diseased misshapen berries will detract from the appearance of the bunch, thus reducing its value in the market. Cracking of the fruit is sometimes followed by rotting because of other fungi.

In South Africa and South America some extremely high losses have been reported. For example, in the epidemic year 1950 to 1951 in Chile 80 to 100 per cent of the crop was lost on several hundred acres of a susceptible variety, which constitutes about 90 per cent of the grapes grown in this region.

Causal Organism. De Bary in 1874 accurately described the disease and the causative fungus, which he named *Sphaceloma ampelinum.* This name was retained for the imperfect stage of the fungus by most writers until the discovery of the perfect stage by Shear in 1929, when it was named *Elsinoë ampelina* (DeB.) Shear.[1] *Gloeosporium ampelophagum* Sacc. was another name used for the imperfect stage.

In many respects this fungus is very similar to the raspberry anthracnose fungus *E. veneta.* The *acervuli* in the center of the lesions produce heaped up, pink masses of gelatinous conidia. The *conidia* are one-celled, hyaline, oblong or ovoid, 5 to 6 by 2 to 3 μ. The conidia are produced throughout the growing season, but only during periods of wet weather when the canes are soaked.

The perfect stage is found in the spring in the cankers on the previous year's growth or on pruned canes which have been left on the ground. The *ascocarps* are small, inconspicuous black bodies in which are embedded scattered, globular *asci.* The *ascospores* are hyaline, three-septate, and 15 to 16 by 4 to 4.5 μ. Since there is no ostiole these spores must be released by the softening or disintegration of the ascocarp. It is probable that they play little part in initial infection.

Disease Cycle. The fungus mycelium overwinters in the canes and gives rise in the spring to either conidia or, more rarely, ascospores. The conidia are washed on to the developing green tissue where they germinate and penetrate the unwounded shoots. The course of the hyphae has not been accurately determined, but it is known to spread rather slowly through the outer cortical area of the shoot and does not extend deeply. New crops of conidia are produced, and these bring about infection of the fruit, usually after it is half-grown. Tendrils, fruit stems, leaves, and petioles are also infected. Infection may continue throughout the growing season but generally is confined to wet periods during the spring and early summer. On the berry the fungus does not penetrate deeply in the flesh, and often no conidia are produced on the fruit. During rainy periods, however, abundant pustules may develop near the center of the spot, and spore masses are exuded as on the shoots.

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[1] Class Ascomycetes, Order Myrangiales, Family Myrangiaceae.

Hosts. Grape anthracnose is known to occur only on species of *Vitis*. It has been reported on most species of this genus. Of the cultivated grapes both American and vinifera grapes are susceptible.

Varietal Resistance. Concord, Delaware, Moore's Early, and Niagara of the American varieties are reported as resistant, while Champion, Catawaba, Diamond, Norton, and Campbell Early are susceptible. Concord, fortunately, is quite resistant and rarely suffers serious injury.

A number of susceptibility lists of vinifera varieties have been published. Wide differences from immunity to very high susceptibility are evident when these lists are consulted. In South Africa, for example, Du Plessis lists Muscadel, Sultana, and Waltham Cross as highly susceptible, while Ferdinand de Lesseps and Golden Queen are resistant, and Gros Colman, Raisin Blanc, and Stein seldom if ever show traces of the disease. In Chile, Negra Pais, which constitutes 90 per cent of the acreage, is very susceptible. In general these lists do not differentiate between vine and berry susceptibility.

Climatic Relations. All who have had experience with anthracnose agree that rainfall and humidity are the most important climatic factors. A region having light total rainfall as in California is seldom troubled with this disease. Even in regions where annual rainfall is high, the disease may be severe only in those years of prolonged rains during the spring and early summer months. Thorough wetting of the lesions is necessary to start spore production, and distribution is dependent mainly on washing rains.

Control. In regions where anthracnose is a serious problem a new vineyard should be planted to resistant varieties if these are available and meet market demands.

Since most of the previous season's shoot growth is removed in pruning, it is often possible to eliminate practically all the canes showing cankers without seriously reducing the number of buds for spring growth. Care should be taken to remove shoots which are likely to trail near the ground.

Spraying has proved effective in eliminating the disease, but special sprays are rarely needed if Bordeaux mixture or other fungicides are applied for black rot and downy mildew control. In South Africa a dormant spray of copper sulfate without summer sprays was found to be fairly effective. Spring sprays if used should be applied first when the new shoots are 7 to 8 in. long. The number of subsequent sprays will depend on weather conditions.

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Dead Arm of Grapes

Various common names have been applied to this disease, among others "necrosis," "side arm," and "black knot." The term "dead arm" is now generally accepted as most descriptive.

History and Geographic Distribution. For a number of years after the discovery of the true nature of the disease by Reddick in 1909, dead arm was regarded as of importance only in northeastern United States. In 1928 Coleman called attention to the serious loss of vines in the Ontario region of Canada and gave additional details on the penetration and progress of the fungus in the host tissues. Later dead arm was reported from California on vinifera grapes, and in 1949 it was regarded as "alarming" in South Africa. It is now known to occur in most countries where grapes are grown.

The killing of the entire vine or single arms is the most damaging aspect of the disease. Cases are on record of from 25 to 50 per cent of the vines removed or so badly diseased as to be unproductive. The disease on the berries is not considered of much importance.

Symptoms. As the name indicates, the most conspicuous symptom is the appearance in the spring of "arms" which fail to leaf out or produce dwarfed yellowish clusters of leaves (Fig. 80). Sometimes these are confined to one or two arms, but some vines may be killed back to the ground line, in which case suckers arise from the base of the trunk. On some weakened arms showing yellow crimpled leaves in the spring, new normal leaves develop which mask the diseased condition.

A close examination of the canes shows the presence of reddish-brown spots on the green shoots, petioles, and leaf vines. When these lesions are deep and continuous, the cane may be split. The disease also occurs on the berries with symptoms very similar to black rot.

Actual death of the vines or arms evidently occurs during the winter months.

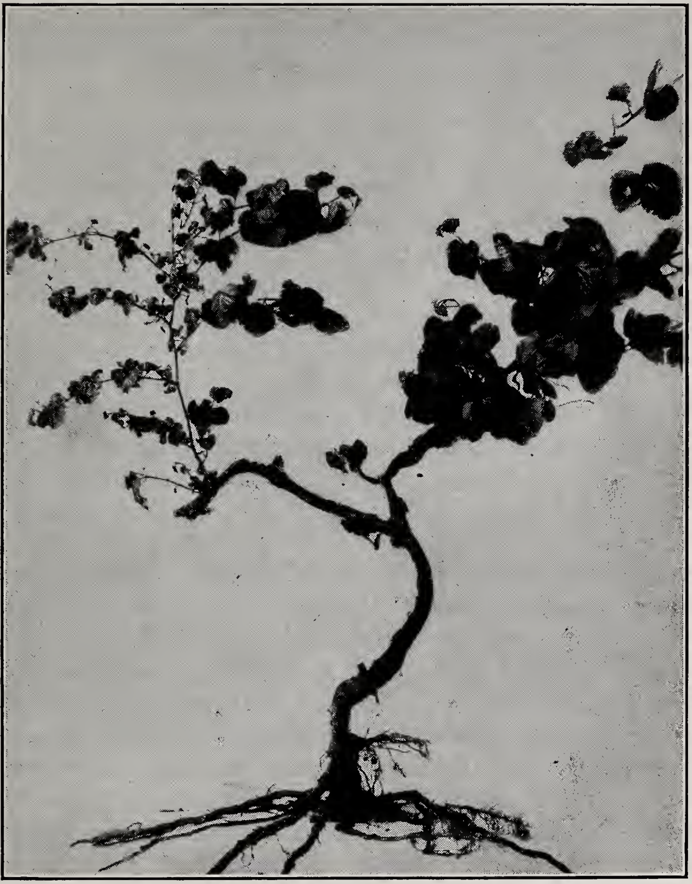

Fig. 80. Dead arm of grape, showing the development of small cupped yellow leaves from the diseased arms. (*Cornell Univ. Agr. Expt. Sta.*)

Causal Organism. The disease is caused by a fungus *Cryptosporella viticola* Shear.[1] Before the perfect stage of this Ascomycete was discovered by Shear in 1911, Reddick had named the imperfect stage *Fusicoccum viticolum.* The perfect stage seems to be rare and of little importance since the fungus overwinters in the host tissue and produces abundant pycnidia in the spring. The *perithecia* are found in stroma buried in the bark of old lesions. The *ascospores* are single-celled, hyaline, subelliptic, 11 to 15 by 4 to 6 μ. The black *pycnidia* are present in

[1] Class Ascomycetes, Order Sphaeriales, Family Sphaerioidaceae.

great abundance on 1- or 2-year-old lesions or on the wrinkled fruit. The inner wall of the pycnidium is convoluted so that the chamber is irregular in outline. The pycnidia are formed in the fall, but the spores ordinarily do not exude until spring. When soaked the spores exude in reddish-yellow balls or tendrils. Two types of spores are found in this mass, one the regular slightly curved elliptic to oblong, hyaline, one-celled spores, and the other elongated filamentous, often hooked structures, called *scolecospores*, which resemble paraphyses of the Ascomycetes. The true *pycnospores* measure 6 to 11 by 1.5 to 2.8 μ.

Disease Cycle. Exudation of the spores takes place in May and June during wet periods. During dry periods the spore masses dry out and become very firm. When water is added the mass swells and spores are freed and dispersed. They are disseminated by washing and are carried to the growing shoots, leaves, and fruit branches. In spite of the millions of spores produced, it seems that only under conditions of prolonged rainy weather, fogs, or heavy dews are they successful in bringing about infection. Reddick states that production of spores is limited to a fairly short period in late May and June and that no spores are to be found the latter part of the summer.

The period between infection and the first appearance of the disease is about 1 month, indicating a slow growth of the fungus in the host tissues. This is also true of the future advance of the fungus after it has become established.

The small dark purple to almost black spots indicating successful invasion are usually confined to the basal area of the cane represented by the first three or four internodes.

Infection also takes place through pruning wounds or accidental mechanical injuries. This accounts for the deep-seated cankers which are found around stubs. The fungus after gaining entrance grows through both phloem and wood. The hyphae penetrate the cells of the phloem parenchyma and the sieve tubes. In the wood the cells of the medullary rays are invaded and deprived of starch. Collapse of the invaded tissue in both wood and phloem results in cracking and pitting, which is a characteristic of the disease. A cross section of the stem often shows a decayed condition resembling "heart rot" of woody plants. The advance of the fungus up and down the branch or stem is rather slow, and the disease may be present for a year or more before the evident dwarfed yellow foliage shows in the spring. Furthermore, the complete girdling of the cane may require even a longer period. Often the cankers on the old stem are not revealed until the outer scaly bark is removed.

Control. Where the disease has become established in a vineyard, cutting out diseased portions of the vine should be practiced. If the entire vine shows symptoms, it should be cut off near the ground line to allow new shoots to develop. Before cutting out diseased shoots or arms, the loose bark should be removed for some distance below the evidence of the disease to locate any cankers along the stem, and cuts should be made well beyond the lowest canker found.

Since most infection takes place through the pruning wounds these should be protected during the period when spores are exuding, which is in May in the North Temperate Zone. This may be done by the use of a good fungicidal spray such as Bordeaux mixture.

In California a dinitro spray, DN-289, applied in the late dormant season gave good control and was less hazardous than the previously recommended sodium arsenite spray.

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Bacterial Blight of Grape

History. Bacterial blight of the vine was first described by Baccarini in 1893 as causing a serious disease in Italy which he called "malnero." He isolated a bacterium from the diseased material and named it *Bacillus vitivorus.* At that time malnero was considered a very destructive disease, especially in Sicily, but while mentioned by later workers it seems to have become of decreasing importance over the next twenty years. The term malnero was later applied to a condition of the vine which was not associated with bacteria. The common name in South Africa is "vlamsiekte."

About 1936 Du Plessis began a study of this disease in South Africa and issued an extensive report in 1940. It was found to be extensive and destructive in the western Cape Province. In 1950 Du Plessis issued another report in which he called attention to the spread of the disease throughout the grape-growing areas of South Africa.

Geographic Distribution. There is evidence that in the early part of the present century bacterial blight was prevalent in Portugal, Spain, France, Italy, and Switzerland. In South Africa it has become general in the grape-growing sections. It has been reported also from Tunis, Canary Islands, Yugoslavia, Argentina, and Austria. It has not been reported from North America. The disease was evidently introduced into South Africa on vines obtained from European countries.

Symptoms. Blight develops on the young *shoots* as pale, yellowish-green, water-soaked spots which darken to grayish black. These extend as stripes along one side of the shoot. The stripes widen around the nodes and often extend into the leaf petioles, side shoots, and fruit stems, which originate from the side of the shoot along which the discoloration has developed. Longitudinal cracks appear on the surface of the lesions as a result of the collapse of the tissue beneath and the increased growth of the living cells along the margin of the streak. The affected shoots are severely malformed by the end of the season. The growth of the developing shoots is retarded, and the leaves are reddish yellow or bright brown. These discolored leaves, yellowish on white varieties and reddish on red varieties, are the most distinctive characteristic of the disease. Blighting and die back occur when the shoots are extensively invaded. Direct infection on the *leaves* either through wounds or hydathodes results in a marginal bright red coloration with final necrosis. These primary leaf infections extend downward into the veins. Thus leaf infection and death may originate by direct infection or by the migration of the bacteria upward through the petioles. Collapse of the leaf may result from cutting off of the movement of sap through the petiolar tissues.

The bacteria exude under moist conditions, and a glistening film is found on the surface of the spots through the drying of this exudate.

The invasion of the *bunch stems* results in a rather sudden collapse and withering of the fruit on these stalks. If the infections occur early, the blossoms and the pedicels on which they are borne are blighted and the bacteria may progress down the pedicels into the main stalk or into adjacent pedicels. Primary infection on the fruit bunch may occur through wounds, as in the case of the leaf. Invasion of the wood results in the formation of gum in the vessels, but external gumming is not evident. Bacterial blight may be confused with anthracnose and the dead arm disease, but a study of the symptoms of these two diseases (page 378 and page 382) will reveal definite macroscopic differences.

Economic Importance. Vines on which bacterial blight is present become practically worthless through the stunting effect on the shoots and the whole vine. If the vine remains alive, the invasion and withering of the fruit on the bunches reduce the yield and value of the crop.

In Europe the disease has practically disappeared from many areas and is regarded of minor economic importance. The loss is still great in South Africa, and here bacterial blight is regarded as the most important disease of the grape. As a result of efforts to check its spread, thousands of infected vines in the producing vineyards have been destroyed and an extensive nursery inspection and certification have been established. Fortunately, only a few varieties grown in South Africa seem to be especially susceptible, thus reducing the loss in productive vineyards planted to resistant varieties.

Causal Organism. Bacterial blight is caused by *Erwinia vitivora* (Bacc.) Du P.[1] It was originally described in 1894 by Baccarini as *Bacillus vitivorus*. It is rod-shaped, capsulate, non-spore-forming, motile by peritrichous flagella, 0.4 to 1 by 1 to 2 μ. It is Gram-negative and not acid fast. The colonies on nutrient agar are light to orange yellow or brown.

Hosts. This disease is known to occur only on the European grape *Vitis vinifera* and some of its hybrids.

Disease Cycle. Infection through unwounded surfaces occurs only on the leaf. On the shoots infections are through wounds, and here only when the bacteria are able to penetrate to the xylem. Young portions of the shoots are more readily infected than older; thus new lesions appear most often on the upper four or five internodes. After infection the bacteria live and migrate within the vine from year to year until death occurs. During the dormant season the bacteria are quiescent, but near the end of the dormant period with the start of sap flow they become very active. The bacteria migrate almost exclusively through the vessels of the xylem. The effect of their invasion, however, is on the cells surrounding the vessels. These at first enlarge and later collapse, so that the sunken streaks and cracks become evident on the surface along the line of invasion. This effect may extend into the pith tissues. The bacterial migration extends upward very rapidly and invades the developing shoots, bunch stalks, and leaf petioles. The invaded vessels show an abnormal gum formation and an increase of tyloses over healthy tissues. This process of invasion of new growth continues in the following and succeeding years. Downward migration is slow.

The spread of the disease in a vineyard is fairly slow. This is due to the fact that infection is rare except through wounds that expose the vascular tissues. The bacteria ooze to the surface of the leaves and on shoot lesions during damp weather and may be carried by various agents to exposed wounds. These wounds may result from pruning operations, breaking of shoots by wind, injury to leaf surfaces and shoots by hail, or injury to the shoots or branches during cultivation, topping, or tying. The main agent of dissemination is probably the workers in the vineyard. This is especially true in pruning and grafting. Diseased grafts, of course, would carry the bacteria when grafted on to a healthy rootstock. Bacteria would be carried on the tools of pruners or toppers working in a vineyard where the disease is present.

Recent investigation in South Africa by MacFarlane showed that the blight bacteria could be isolated from the soil, from 1-year-old prunings and from grape skins and residues from wine factories. She also proved that invasion of the roots may occur in infested soil. These results are significant in relation to the spread of the disease and its control, although other sources of infection probably predominate.

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[1] Class Schizomycetes, Order Eubacteriales, Family Enterobacteriaceae.

Experience both in Europe and South Africa indicates that the disease is more severe in vineyards or areas of the vineyard where soils have a high water-retaining capacity or where drainage is such that the soils remain excessively moist during long periods. High humidity and heavy rainfalls are conducive to increased infection, probably through bringing about oozing of the bacteria and by opening up the shoot tissues through cracking of the bark.

Control. There is no practical control of the disease once the vine becomes infected. Thus control consists in preventing infection on healthy vines. As indicated above, the spread of the disease from vine to vine is largely through man's manipulations. All diseased vines should be removed as soon as discovered. This will prevent careless workers from carrying the inoculum on tools. Regardless of whether or not the disease is known to be present, all prunings, broken branches, and debris in general should be removed and burned. Grape pomace or other wastes of wineries should not be used as mulch in the vineyards.

Fortunately, care in obtaining propagating wood from vines certified to be free of the disease is effective in reducing the chances of infections in areas relatively free of the disease. Even in plantings in areas where the disease is established, the use of clean vines and proper sanitary measures will slow up, if not eliminate, the spread of the disease.

Spraying for the control of bacterial blight alone is of little value but may aid in the control of certain fungi which constantly appear in the infected canes and cause additional injury.

While varietal difference in resistance is marked, the selection of varieties should be governed by the market demand and the general adaptability of the varieties to the region involved. Naturally, in planting a new vineyard, the grower should seek advice as to whether choice resistant varieties are available.

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Virus Diseases of Grape

Comparatively few virus diseases are known on the grape. Numerous diseased conditions, especially in Europe, have been suspected of being caused by viruses, but in most cases these lack experimental proof. The most important definitely proved virus disease is the Pierce's disease in the European grape vineyards of California. This vine disease has also been called "Anaheim disease" and "California vine disease."

History of Pierce's Disease. European grapes from Spain were brought to Mexico as early as 1525 on the order of Cortez. These were mainly of the Mission variety, so named because they were planted around the numerous missions in "New Spain." On the establishment of a number of missions in southern California between 1775 and 1800, Mission vines were planted in extensive vineyards around these missions. By 1850 this variety, together with a few other European varieties, had become established in vineyards throughout the coast region around the cities of Los Angeles and Santa Barbara. One of the notable plantings was about the little town of Anaheim, southeast of Los Angeles, where a group of German settlers had planted extensive vineyards around 1858. It was in these vineyards that the disease was first noted. Pierce, a special agent of the U.S. Department of Agriculture, was sent to this region shortly after the first outbreak of this devastating disease. His report, published in 1892, contains the results of a very extensive investigation of the first appearance of the disease. He was able to establish the fact that neither this disease nor any similar one had been known over the two hundred years of grape culture in California until the outbreak in 1884–1885. His thorough documentation of the situation at Anaheim clearly established 1884 as the first appearance of the disease. The effect of the disease is so striking that there can be little question but that it was an entirely new malady. During this epidemic extending from 1884 to the late 1890's, the disease spread into neighboring counties of San Diego, Riverside, San Bernardino, Ventura, and Santa Barbara. Many of the vineyards were wiped out and were replaced by citrus fruits. It destroyed more than 35,000 acres and caused the closing down of the local wineries. The extensive vineyards in the San Joaquin Valley were not affected during this period. The disease appeared to be quiescent from about 1900 to 1920. Between 1920 to 1930 the disease appeared in the grape-growing region of the San Joaquin Valley, and by 1935 it was realized that a new epidemic had erupted. This epidemic continued with increasing losses until about 1941, when it declined rather abruptly in the San Joaquin Valley. In other parts of southern California some outbreaks occurred during this period but were more local.

The second epidemic of Pierce's disease initiated an extensive renewed investigation of the nature of the causal agent and methods of transmission. These investigations were undertaken by a group of plant pathologists and entomologists of the California Agriculture Experiment Station and the California State Department of Agriculture. Hewitt in 1939 announced the successful transmission of the disease to healthy vines by root grafting and by budding. In 1942 Hewitt, Frazier, and Houston reported successful transmission by leaf hoppers and suggested the possible connection between Pierce's disease on grape and a virus disease of alfalfa called alfalfa dwarf.

With the discovery of the cause of the disease, rapid progress was made in the period from 1940 to 1950. The results of these investigations were published in a series of papers which established the following important facts.

1. Natural transmission was mainly by three sharpshooter leaf hoppers, *Carneocephala fulgida*, *Draeculacephala minerva*, and *Hordina circellata*. In addition, it was determined that other species of leaf hoppers and spittle insects were able to transmit the virus.
2. It was definitely established that alfalfa dwarf and Pierce's disease were caused by the same virus.
3. Freitag determined that many host plants aside from grape and alfalfa might be carriers of the disease. He stated that 111 species in 41 families were carriers. Many of those were symptomless carriers.
4. The host range, feeding habits, and life cycle of the principal vectors were studied, thus giving a clue as to the failure of some of the earlier recommended control measures. Surveys were made during this period under the direction of Stout of the State Department of Agriculture to determine the extent and severity of the disease in the various grape-growing areas of California.

Geographic Distribution. Until 1953 Pierce's disease was known to occur only in California with a single case in a Thompson Seedling vineyard near Carrizo Springs, Texas. Stoner in 1953 reported experiments which demonstrated that the long-recognized degeneration of grapes in Florida was caused by a transmissible virus which produced symptoms on grape identical with Pierce's disease and on alfalfa identical with alfalfa dwarf. A similar disease has been reported from Argentina.

Economic Importance. The devastating effect of Pierce's disease is illustrated by the records from the epidemic of 1884 to 1900, in the region around Los Angeles. During the next few years following the 1884 appearance of the disease, some 35,000 acres of productive vineyards were destroyed. This not only involved the loss of vineyards but ruined the local wineries and disrupted all the business activities of the communities which depended largely on the grape industry. Vines are usually killed in from 1 to 3 years after infection. Any fruit produced after the first year is worthless. In the first epidemic many of the Mission vines were from 20 to 50 years of age and were producing 8 to 10 tons per acre. Within 2 years the production dropped to 2 tons. Subsequent attempts to replant the area were unsuccessful. The losses in the lower San Joaquin Valley in the second epidemic were similar to those reported in the first epidemic. Many vineyards were rendered so unproductive that they were abandoned. Attempts over a century to grow grapes in Florida have always resulted in failure. Some varieties flourish for a short period and then die out or become unproductive. This "degeneration" has been ascribed to many different factors, but if one accepts the Pierce's virus as the main factor, it may be reasoned that here is a case where a financial loss results from the failure to establish a crop otherwise suitable to the region.

Symptoms. Symptoms may vary some with the variety and locality concerned, but are fairly uniform in pattern. They may be summarized as follows:

> (1) Scalding, a progressive drying of the leaf tissues while still green, usually from the margin toward the leaf petiole, followed by browning of the scalded areas; (2) dwarfing, wilting, withering, drying, and, in late season, premature coloring of the berries on part or over the entire vine; (3) delayed foliation of a part or of the entire vine in the spring; (4) interveinal chlorotic mottling and sometimes deforming of the first 2 to 8 leaves of a shoot; (5) dwarfing of shoot growth on a part or over the entire vine; (6) failure of the canes to mature evenly; (7) gradual dying of the root system; (8) symptoms often show variation with age, variety, and locality, dying of vines from 3 months to 4 or 5 years (*Hilgardia,* **19**:210–211 (1949)).

Under California conditions the scalding and burning of the leaves, the first symptoms, may occur any time after mid-June. In the second season delayed growth and dwarfing may be observed. This is followed by leaf scalding and later defoliation, starting at the base of the shoot. Death of the roots soon follows. Symptoms similar to some of those listed above may be the result of other factors, but such cases are readily eliminated by careful observation.

Internally, the disease is characterized by the browning of the xylem (wood). An increase of tyloses and gumming in the vessels accounts for this browning.

Alfalfa dwarf caused by the same virus is characterized by stunted *plants*, with short stems and small leaves. After each cutting the stems become shorter, are fewer in number, and the leaves smaller. There is no leaf mottling. The vessels in the xylem of the alfalfa roots also show the characteristic gumming and tylose formation, giving a yellowish cast to the infected roots. The presence of alfalfa dwarf in a grape-growing region is often helpful in locating Pierce's disease.

Causal Agent. The virus causing Pierce's disease has been given the scientific name *Morsus suffodiens* Holmes. It is of the xylem-invading type.

Hosts. This virus has proved to have a wide range of hosts, most of which are symptomless carriers. Transmission experiments have proved that 111 species in 41 families are carriers. Alfalfa, species of clover, and the snowberry plant (*Symphoricarpos albus*) show definite symptoms and are consequently useful in transmission studies. All species and varieties of *Vitis* are susceptible so far as tested.

Transmission. The virus has not been successfully transmitted by mechanical means, such as rubbing or injection of infective juice. It is not seed-borne. The virus has been transmitted by grafting pieces of roots, trunks, or canes from diseased to healthy vines. In California three sharpshooter leaf hoppers are regarded as the most important insects in the natural spread of the disease. The blue-green sharpshooter *Hordina circellata* is regarded as the most important vector, especially in the vineyards of the coastal regions. The green sharpshooter *Draeculacephala minerva* and the red-headed sharpshooter *Carneocephala fuligida* also are of importance, since they are commonly found in the vineyards, although the grape is not a favored host of the latter. In Florida the yellow-headed sharpshooter *C. flaviceps* was found to be effective in transmission studies and to be abundant in the experimental grape vineyard. Bermudagrass seemed to be its principal host. In addition to these insects 20 species of leaf hoppers and four species of spittle bugs are known to be able to transmit the virus to grapevines.

The virus is transmitted only when the insects feed on the xylem tissues of the diseased plant and viruliferous insects must penetrate the xylem tissue of the healthy vine in order to bring about infection.

The virus is limited to the wood of the grape. It develops in the parenchyma cells of the xylem, since these are the only living cells. The main effect, however, is on the vessels, in which gum and tyloses are produced in such abundance as seriously to restrict water movement and thus bring about the drying, wilting, and stunting of leaves and shoots. Gumming also occurs in the phloem and the leaf mesophyll of inoculated plants, even before external symptoms are evident. The virus moves both up and down from the point of inoculation, but the movement is comparatively slow.

The incubation period from the time of inoculation to the appearance of symptoms in the vine is from 8 weeks to 15 months, depending on the time of inoculation and the growth conditions of the plant.

Little is known about the physical character of the virus.

The relation of climatic factors to Pierce's disease are not well understood. It appears that rainfall has some influence, since the two periods during which the disease was most prevalent were characterized by 1 or more years of abnormally high rainfall in the regions where the greatest loss of vines occurred. It is probable that this is related to the production of more succulent growth of host plants of the vectors. On the other hand, hot dry periods are conducive to more rapid development of symptoms and injury on infected plants.

Control. No adequate control measures have been developed for Pierce's disease. While some varieties appear to be more tolerant of the disease, all are susceptible, including the native American species.

Extensive experiments in California proved that vineyards where the diseased vines were systematically rogued developed about the same proportion of diseased vines as unrogued ones. Spraying for the destruction of vectors also failed to reduce the new infections. Weed control in the vineyard and neighboring areas gave little hope of control.

Cuttings from diseased vines carry the virus; thus the grower should be certain that cuttings for propagations are obtained from vineyards where the disease is not known to occur.

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Grape Virus Diseases of Minor Importance

Three other virus or viruslike diseases have been reported from California.

"White Emperor" is the common name of a disease which results in the production of greenish yellow or pink fruit on the Emperor variety which normally produces bright red berries. The fruit is low in sugar content, although about normal in size. The leaves early in the season are darker and thicker than normal, are wrinkled, and curl down at the margins. Later they turn to a reddish or bronze color along the main veins and yellowish between the veins. Grafting experiments proved that when wood from the white Emperor is grafted on healthy vines, these produce the light-colored fruit. The disease does not appear to spread in the vineyard, but the transmission experiments indicate that it is of virus origin. It is not known to occur outside of California or on any variety *other* than the Emperor. Care in the selection of propagating wood will control this disease.

Vine mosaic reported in 1945 from California appears to be the same as a virus disease called *panachure* in France. Under this name it has been reported from several European countries and from South Africa. This disease is characterized by the development of bright yellow leaves on the spring growth. Some of these become chlorotic as they age, others develop some green color. Leaves produced later in the season may appear almost normal or with some mottling and chlorosis. Diseased vines of some varieties fail to set fruit.

The virus can be transmitted by grafting, but no insect transmission has been recorded. It is reported to show little if any spread in the vineyards where it occurs. For this reason control consists in selection of propagating wood from healthy vines. Since diseased vines are of no value they should be removed.

Fan leaf is the third minor virus disease in California. It was reported in 1948 on the variety Pinot Chardonnay in the Santa Clara Valley and has since been found on a number of varieties. Hewitt considers it the same as one occurring in Europe under various common names. Like fan leaf and vine mosaic, the European virus does not appear to spread in the vineyard but can be transmitted by grafting.

The symptoms are "gradual dwarfing of the vine; early-season dwarfing of shoots; deformity of shoots and leaves; mottling of leaves on early-season growth and shelling of flowers from the clusters, or the setting of almost seedless berries" (Hewitt).

The fan leaf character of this disease is similar in many respects to that produced by some weed killers, such as 2-4-D, to which the grape is exceptionally sensitive. It is easily distinguished from this condition by several definite differences: Leaves injured by 2-4-D are fan-shaped with the narrow sharp-pointed serrations, but the outline is more even with no deep cuts or twisting as in fan leaf; the white veins and veinlets are much more evident; no dwarfing of the vine occurs, and the injured leaves appear late in the season on shoots of normal length; unless the

2-4-D injury is severe there is no shelling of the blossoms or effect on the berries.

Control measures are the same as for the other minor virus diseases.

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Some European Virus Diseases

A number of abnormal conditions of grapevines have been studied in European countries during the past sixty years. Diligent research failed to reveal any insect, bacterium, or fungus responsible for the condition. Nutritional and climatic disorders were fairly well understood, but these factors could not account for some other vine conditions which were met with throughout the grape regions of Continental Europe. These disorders were given various local names based on the prominent symptoms. This led to considerable confusion as to the identity of the various diseases. It was natural to suspect that some at least of these diseases might be of virus origin. Transmission of some of the diseases proved that this assumption was warranted. Variations in symptom expression in different geographic regions, together with variations due to varietal differences, caused some controversy as to proper classification of the diseases, that is, whether one or several or a mixture of viruses were concerned.

In 1950 Du Plessis, after reviewing the literature and visiting the European grape-growing regions, attempted to clarify and reconcile the diverse opinions in regard to these diseases. He had also visited the grape-growing regions in North America and was thus able to compare the European and American viruslike diseases. The account which follows is based largely on his publication. He classifies the diseases as follows:

1. Pierce's disease, occurring in California and the Argentine (also in Florida)
2. Roncet, urticado, nettle leaf, or Reisigkrankheit, occurring in California, Portugal, Spain, France, Switzerland, Italy, Germany, and probably in several other European countries (called "fan leaf" by Hewitt in California)
3. Panachure, or white mosaic, occurring in California, Portugal, Spain, France, Switzerland, Italy, South Africa, and according to reports also in Bulgaria and Czechoslovakia (called "vine mosaic" by Hewitt)
4. True mosaic in France, Switzerland, and Italy

5. Leaf roll, or Rollerkrankheit, occurring in France, Switzerland, and Germany
6. Witches'-broom, occurring in Switzerland
7. Distortion disease, occurring in Sicily

Of the above-listed diseases the one most commonly mentioned in European literature is **roncet** which is generally called "court-noué" in France. This is by far the most common and destructive disease in European vineyards. The symptoms most commonly mentioned are (1) fan-shaped leaves with deep indentations and increased serrations, suggesting the term nettle leaf and (2) shortening of internodes and zigzag staggering of these. Reduction in fruit bearing and general stunting result. The virus has been repeatedly transmitted by grafting. (See under Fan Leaf for further symptoms and effect.)

Panachure, a white mosaic, is the next most common virus disease in European vineyards. The chlorotic spotting is much more pronounced in this than in true mosaic. The shoots are stunted, and fruit production reduced. The virus has been transmitted by grafting in Switzerland. (See under Vine Mosaic.)

True mosaic has not been found in America but is common in France. Leaves infected with true mosaic show light transparent-green spots, grouped or scattered on the leaf blade. Some vein clearing is evident. The leaves retain their normal outline, thus distinguishing it from roncet. The leaves may be dwarfed and somewhat wrinkled when severely infected. Severe stunting of shoot growth results. No information is available on the transmission of the disease, but it is probably of virus origin.

The remaining diseases in the above list, leaf roll, witches'-broom, and distortion disease are of minor importance or restricted in their distribution. Pierce's disease has been discussed in detail (p. 389).

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Diseases of Grapes Incited by Nematodes

In 1954 Raski gave a good account of the nematode situation on grapes in connection with experiments on soil fumigation in California vineyards. The important grape industry of California has been threatened by the premature decline of vines and accompanied decreased productivity, especially in the San Joaquin Valley and southern California. Attempts to replant weakened vines in old vineyards often result in failure of the plants to become established or thrive. Such plants often fail to produce a crop. Whether or not this decline is directly caused by nematode attack and the relative importance of the different species have not been determined.

Root knot is commonly found on grapes in southern grape-growing regions, especially where this crop is grown in light, sandy soil. Until recently, this nematode was classed as *Heterodera marioni*, but Raski reports that two species, *Meloidogyne incognita* var. *acrita* Chitwood and *M. javanica* have been found in California, the former much more frequently than *M. javanica*. For further information on root knot nematodes see Chap. 1 and also under Nematodes on peach.

The **root lesion nematode,** *Pratylenchus vulnus*, according to Raski, appeared to be an equally serious parasite wherever it was found infecting grape roots. While generally found in the vineyards, it was not encountered as frequently as the root knot nematode (*Meloidogyne incognita* var. *acrita*). Another species of root lesion nematode, *P. minyus*, was found quite frequently in soils taken about the roots of grapes. (Information concerning the root lesion nematodes will be found in Chap. 1 and also under Nematodes on peach.)

Control. Raski ran a series of field experiments in California on the control of nematodes by soil fumigation. The materials used were ethylene dibromide (EDB) and 1,3-dichloropropene—1,2-dichloropropane mixture (D-D). He concluded that while in some cases great improvement was evident in the growth of the vines, in other cases no significant difference could be demonstrated. In making nematode counts in one vineyard he found that the nematodes and injury varied greatly in different parts of the vineyard regardless of the treatment.

Crop rotation, using crops resistant to nematode attack, seems at present the most practical method of control. Where old, weakened vines are removed, the area should be planted to a resistant cover crop for at least 3 years before resetting to grape.

Lider, in 1954, investigated the resistance of grape rootstocks to the root knot nematode (*M. incognita* var. *acrita*). He reported that nematode-resistant rootstocks have been in commercial use in California vineyards for some years. The clone (*Vitis solonis* × Othello) has been one of the most popular. Rootstocks of *V. champini* have shown promise in experimental plantings and are now being used on a limited commercial scale. Varieties grafted to these stocks have not been entirely satisfactory either because of poorer fruit quality or lower yield. *V. vinifera* and *V. labrusca* rootstocks are quite susceptible to attack by *M. incognita* var. *acrita*, but several American species are quite resistant. These include *V. champini*, *V. solonis*, *V. doaniana*, *V. cinerea*, and *V. candicans*. While an entirely satisfactory rootstock has not yet been obtained, it is hoped that hybridization will soon result in an immune variety which will give satisfactory growth when fruiting varieties are grafted on its seedlings.

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CHAPTER 10

DISEASES OF STRAWBERRIES

Strawberries are the favored fruit of many nations of the world. This fruit may be found in the markets from the tropics to the polar zones in both hemispheres. Species of strawberries are native in most regions of the Temperate Zones, but the large-fruited, productive varieties now cultivated are derived from chance crossing of two species, both native to the Americas. A Chilean species (*Fragaria chiloensis*) was introduced into France in 1712 and a little later became established in English gardens, where the North American species (*F. virginiana*) was being grown. It is probable that a chance cross of these two species in England gave rise to our modern varieties. It is known that seedlings from such crosses often give rise to superior, large-fruited varieties. Plants of some of these superior English varieties were brought back to North America to furnish the foundation for our commercial varieties.

The strawberry belongs in the genus *Fragaria* of the rose family (*Rosaceae*). The berry is derived from the swollen receptacle which is studded with the single-carpel pistils which become the "seeds" of the fruit. The toothed calyx, which subtends the fleshy berry, usually remains green until the fruit is harvested and is picked with the fruit.

The strawberry plant, after producing the fruit, sends out a number of runners along which new plants arise at intervals. A single mother plant in a vigorous condition may produce from 25 to 50 new plants. These become the bearing plants of the next season or are dug and planted in a new bed to produce matted rows for the crop of the succeeding seasons. Mother plants will form new crowns and roots and produce berries for a number of years, but these are usually inferior to the younger plants formed from the runners.

Since the strawberry has a limited season, this fruit is often sent to distant markets and thus gives rise to transportation problems common to shipped perishable fruit.

Leaf Spot of Strawberry

"Common leaf spot," "leaf blight," and "rust" are the only common names which have been generally applied by growers to this disease.

"Rust" was more generally used in the early history of the disease in eastern America, while "leaf spot" or the "spot disease" is more frequently heard in recent years. Leaf blight is now applied to a disease caused by a different fungus.

History. Strawberry leaf spot was described as early as 1863 in Europe. It did not attract much attention in America until between 1880 and 1890, but there is evidence that it was known long before this time and was probably indigenous to America on the parent wild species. In 1878 "rust" was mentioned in the *Transactions of the Illinois State Horticultural Society* as though it were a commonly known disease, and certain varieties were listed as being especially susceptible. Strawberry-growers soon learned to avoid varieties which showed excessive susceptibility to leaf spot, and breeders learned that it was not a good policy to release such varieties. As a consequence, there has been a selection for resistance throughout the years, although breeders were more interested in other desirable characters and did not select primarily for disease resistance.

Geographic Distribution. Leaf spot has been reported from all sections of the world where the strawberry is grown. Since the introduction of strawberries into new countries was by the transportation of runner plants, it is not strange that the disease, even if originally confined to one country, would soon become world-wide in its distribution.

Economic Importance. Leaf spot is of great importance where the temperature and moisture are favorable for severe epidemics. The loss consists of a decided reduction in the total yield, reduction in the grade of harvested berries, and a weakening of runner plants, which in turn would be reflected in the yield the following season. Where highly resistant varieties can be grown and where conditions for infection are not normally favorable, the loss is not great. However, in some cases inferior market varieties are grown because of their resistance to leaf spot, and thus indirect loss results. The most accurate results on crop loss are reported by Plakidas, from Louisiana, where the disease may be very severe. Leaf spot was controlled by the use of Bordeaux sprays. One season's results showed an increase of 107¼ crates per acre on the sprayed plots, which, at the average price of $2.50 per crate that season, would have yielded $268.12, or, after deducting all expenses, a net profit of $158.12 per acre. Another year's results showed a net profit of $315 per acre in favor of the sprayed plots.

Symptoms. Leaf spot is most frequently evident on the blades of the *leaflets*, but may appear on the *petioles*, *fruit*, and *fruit stems*. The lesions may be seen first on the upper surface as small, deep-purple, somewhat indefinite areas. As the spot enlarges, the central area becomes brown, but soon turns to a definite white spot in older leaves or to a light brown in young tender leaves. An indefinite dark purple zone surrounds the central light area, giving the whole a bird's-eye effect (Fig. 81). When the infections are bunched on the leaf, the purplish area may become confluent and extend around a number of the white spots, and if the infection is near the edge of the leaflet, the purpling often extends to the border. On the undersurface the symptoms are much the same as on the upper, but the coloring is less intense. Here the prominent veins which are touched by any of the spots take on a reddish-purple color which extends some distance beyond the infected spots.

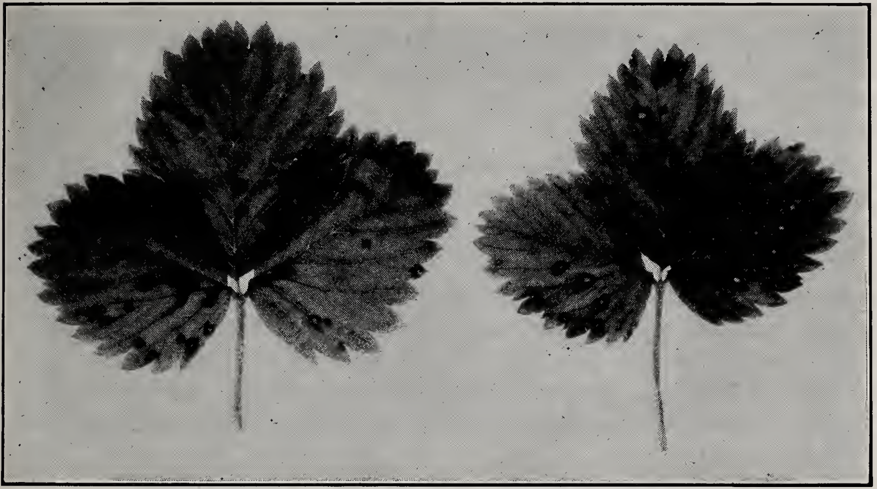

FIG. 81. Strawberry leaf spot. Left, early stage; right, late stage.

The central light area is usually 1 to 3 mm in diameter, while the purple border may be twice as broad. The spots may be few or numerous, scattered or localized. When numerous, they often cause the death of the leaf tissue over a segment of the leaflet, or entire leaflets may be killed. Later in the season the spots become more definite in outline, and dark specks may be observed in the white surface of the central dead area.

Causal Organism. The organism responsible for leaf blight is *Mycosphaerella fragariae* (Tul.) Lindau.[1] The perfect stage was described by the Tulasne brothers in 1863 as a *Stigmatia.* Later this was transferred to the genus *Sphaerella,* and then to *Mycosphaerella.* The imperfect stage was described as *Ramularia tulasnii* by Saccardo in 1879. Peck also described this stage in 1883 under the name *R. fragariae,* evidently under the impression that it was different from the European species.

Hosts. *M. fragariae* has been collected on most species of *Fragaria.* It has also been recorded as occurring on *Potentilla monspeliensis,* a common weed.

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[1] Class Ascomycetes, Order Sphaeriales, Family Mycosphaerellaceae.

Morphology. The perfect stage of this fungus is found at the edge of the leaf spots during the autumn months. The small, black, globose *perithecia* produce few asci. The asci are clavate, eight-spored, and are about 40 μ in length. The two-celled, hyaline, fusiform *ascospores* measure 3 to 4 by 15 to 18 μ. They germinate in the ascus. The *conidia* of the *Ramularia* form are produced in great abundance on short conidiophores which arise in small clusters over the entire undersurface of the diseased area, but are most often conspicuous during damp weather as a downy white growth on the purple area adjoining the light central spot. The conidia are hyaline, cylindrical to ellipsoidal, 3 to 4 by 20 to 40 μ, at first one-celled but later two- to three-celled. Small, black *sclerotial bodies* are also developed on the spots. These, on germination, produce conidiophores and conidia of the *Ramularia* type. A *Septoria*-like pycnidium has been described by various authors and by some is regarded as a spermagonium. The mycelium is intercellular with no evidence of haustoria.

Disease Cycle. The strawberry leaf spot fungus has no difficulty in passing the winter months. The strawberry is perennial, and often a few of the older leaves of the runner plants which have become infected live over winter in the green state. From the infected spots on these a new crop of conidia are readily produced in the spring, at which time the newly developing leaves are susceptible. The sclerotia on dead leaves may also produce a crop of conidia in the spring. It is probable that most primary infection in the spring results from conidial inoculations and that the perfect stage is relatively unimportant, although infection from ascospores may occur.

Stomatal infection seems to be the rule, and since in most varieties the stomata are limited to the lower side of the leaf, the infections all occur on this surface, although the symptoms are first noticed on the upper surface. Infections occur on the developing leaves of the old plants at any time during the spring months when weather conditions are favorable. If the variety is especially susceptible, this infection may be serious enough to restrict the growth of the new leaves and thus not only reduce the crop but also expose the berries to the hot sun. After harvest the runners produce new plants which may escape infection during the hot summer months but are frequently seriously spotted during the rainy early-autumn season. If these plants are mulched before heavy freezes, many of the infected leaves on these as well as on the parent plants remain alive during the winter. The runner plants may be dug to establish new beds the following spring, in which case the fungus is likely to be transferred with the plants. Conidia are produced in great abundance on any infected organ and are readily carried by water and other agencies to healthy plants. Wild plants and escapes from cultivated fields often grow in abundance along fence rows and are usually thoroughly infected.

The incubation period for the leaf spot fungus is from 10 to 14 days. The amount of infection which occurs in a bed is dependent upon a number of factors, but the most important is the prevalence of fairly prolonged damp periods. Apparently, infection takes place throughout the entire growing season, since at all times susceptible tissue is present.

Control. Injury from leaf spot may be reduced to a negligible point by spraying, but it is only under conditions where the disease is a limiting factor in production that spraying is practiced. For the most part, strawberrygrowers depend upon a certain degree of resistance on the part of the varieties which they select. Varieties which "rust" badly are not looked upon with favor and are soon discarded. This has led to a rigid and somewhat unconscious selection for resistance on the part of nurserymen who are interested in the introduction of new varieties. Attempts have been made to determine the factors which constitute resistance. Since the genetic constitution of our cultivated strawberry is known, the relative susceptibility of the two wild parents, *Fragaria virginiana* and *F. chiloensis*, might be an important factor in determining the resistance of the hybrids. It is well known, however, that the parent species in the wild vary greatly in their resistance and that hybrid plants show great variability in this respect regardless of the types selected in crossing. Since many other factors need to be considered in developing a new variety, the factor of resistance must be left more or less to chance. Plakidas has found that the number and location of the stomata are not correlated with resistance.

A number of lists of varieties showing their relative resistance to leaf spot have been prepared in the past, but since growers' preferences for varieties change very rapidly, these lists soon become useless. Plakidas in 1948 reported the existence of strains of *Mycosphaerella fragariae*, thus complicating selection for resistance.

Sanitary measures undoubtedly aid in reducing leaf spot injury. Such measures, however, must be subordinate to good cultural practices. Planting in well-drained soil, keeping down weeds, and care in spacing runner plants in matted-row culture are good cultural practices which increase the rate of drying through air circulation and thus reduce chances of infection. Runner plants should always have the older leaves removed before setting. This practice alone often eliminates the disease during the first cropping season.

In renovating old beds, mowing and burning over the patch is practiced in some sections. While this undoubtedly reduces the inoculum, it is not advocated as a good cultural practice by most horticulturists.

Spraying for leaf spot control has been advocated since 1887, and while satisfactory results have been reported, regular spraying has not become an established practice in America. In southern United States where leaf spot and leaf scorch are limiting factors in production, an 8-8-100 Bordeaux mixture has proved very effective in controlling leaf diseases and in increasing production. The time and number of applications depend upon the nature of the season and the type of culture practiced. Applications at 10-day intervals should be made during the growth period of the bearing plants and during the summer months when the new runner plants are becoming established. Ferbam and Captan have been fairly successful in controlling this leaf spot.

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Strawberry Leaf Scorch

This disease has received little attention until recently, since in the past it was confused by growers in general with the *Mycosphaerella* leaf spot and was commonly called "leaf spot." The name "scorch" was given the disease by Stone in 1922.

History. The disease is known to have occurred in Europe as early as 1832. In America it was collected in Illinois in 1883 and was described from New York in 1884. Herbarium specimens of wild strawberry often show this disease, so that it was evidently of common occurrence on both wild and cultivated strawberries in this country. Like the leaf spot, there is no way of determining the origin of this disease.

Geographic Distribution. Leaf scorch has been reported from all countries where the strawberry is grown and is generally distributed over the United States. In general it seems to be more prevalent in the southern than in the northern states, although it has caused serious injury at various times in New York and Wisconsin.

Losses from scorch result when the disease becomes established early in the season and weather conditions are favorable for its development. The losses consist in reduced efficiency of the diseased leaves; in withering of the calyces which are directly attacked; and in injuries to the fruit stems, resulting in reduced berry size. Losses may be as high as 25 per cent of the crop, but this is exceptional. In regions where the disease is often destructive, certain desirable varieties from other standpoints are avoided. For example, in southern Illinois, when Missionary and Klondike were grown as commercial varieties, the Klondike was so susceptible that it was given up, and Missionary, although not so suitable for that region, was substituted.

Symptoms. Leaf scorch lesions may appear not only on the *leaf blades* but also on the *petioles*, *fruit pedicels*, and on the *sepals* of the calyx. In a very early stage leaf scorch lesions resemble those produced by the leaf spot organism in that small dark purple spots appear scattered over the upper surface of the leaflets. It is not difficult to distinguish the two after the spots have developed. In the mature condition the leaf scorch spots are large and irregular in outline and never show the white central area characteristic of the leaf spot disease (Fig. 82). On the contrary, the black fruiting bodies which develop in the central area give the leaf scorch disease a "tar spot" appearance. Often large areas of the leaflet or the entire leaf take on a reddish or light purple hue quite distinct from that produced by any other strawberry disease. The spots are from 1 to 5 mm in diameter and often are so numerous that they coalesce. Badly infected leaves often curl and "burn." The lesions on the petioles and pedicels are elongated as compared to those on the leaves. On the

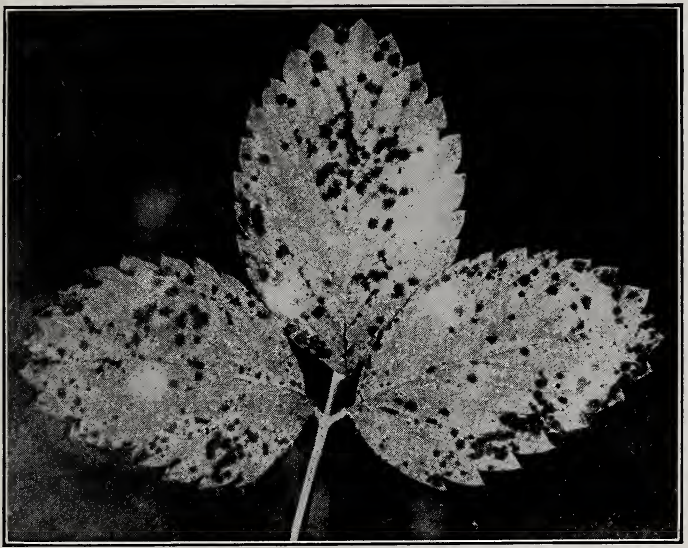

FIG. 82. Strawberry leaf scorch showing indefinite purple borders and central black spot.

calyces the disease results in a rather rapid withering and premature death, thus detracting from the appearance of the harvested fruit.

Causal Organism. Unfortunately, the fungus causing leaf scorch has received an unusual number of scientific names, both genetic and specific. In 1832, the imperfect stage on strawberry was described as *Leptothyrium fragariae,* which in 1849 was transferred to the genus *Gloeosporium.* In 1877, Saccardo described the same organism as *Ascochyta fragariae* on *Fragaria vesca* and *F. chiloensis.* In 1884, Peck in New York described what he considered a new species on *F. virginiana* as *A. colorata* on the basis that it differed in minor characters from that of Saccardo. The character of the fruiting bodies led Saccardo, in 1896, to transfer his species to the genus *Marssonia,* and he renamed it *Marssonia fragariae,* stating that it was synonymous with *M. potentillae* (Desm.) Fisch., but without evidence based on cross inoculations.

The perfect stage of the leaf scorch organism was found by Earle, near Anna, Illinois, in 1883. Ellis and Everhardt named this *Peziza earliana,* in 1884. Saccardo later (1889) transferred this to the genus *Mollisia,* under which name it was known until 1924, when Wolf published the results of a detailed morphological study and, because of the similarity of the organism to that causing black spot of roses (*Diplocarpon rosae* Wolf), transferred it to the genus *Diplocarpon.* Thus the present scientific name is *Diplocarpon earliana* (E. & E.) Wolf.[1]

Stone, in 1919, succeeded in establishing the fact that *M. fragariae* was the imperfect stage of *D.* (*Mollisia*) *earliana.*

Hosts. As stated above, Saccardo and others regarded the *Marssonia* leaf spot on species of *Potentilla* as identical with that on *Fragaria.* There is no evidence that cross inoculations were made until 1924, when Wolf established the fact that the two were distinct by extensive attempts at cross inoculation. The leaf scorch fungus, therefore, is known to occur only on species of *Fragaria* (*F. virginiana, F. viridia,* and cultivated hybrids).

Morphology. In the imperfect stage (*Marssonia*) the *conidia* are produced in *acervuli,* which usually appear on the upper leaf surface as small but conspicuous black, hemispherical, glistening dots, few in number and irregularly distributed over the lesions. As indicated by the confusion in nomenclature, they resemble pycnidia. They are superficial in that they are formed between the outer wall and cuticle of the epidermis. The acervuli are from 0.1 to 0.2 mm in diameter. The *conidiophores* arise from a thin basal stroma as short, cylindrical cells which give rise to successive crops of spores. The abundant spore production pushes up the cuticle which is later ruptured. There is no upper fungal covering, and the impression that the fruiting body was a pycnidium was based on the rather superficial observation of this cuticular covering. The *conidia* are hyaline, asymmetrically two-celled, with the larger, upper cell abruptly curved into a beak. They measure 18 to 30 by 5 to 7 μ, are constricted at the septa, and are guttulate. The ascigerous stage may be found on the lower surface of old lesions in the early spring (March) and show as small black dots visible to the naked eye. They may occur on either leaf surface but are more abundant on the lower. The fruiting body is a true *apothecium,* discoid in shape, from 0.1 to 0.3 mm in diameter. The hymenium arises from the center of a spherical stromalike structure, the upper part of which is forced back by the developing asci and paraphyses. The *asci* are fasciculate, oblong, 55 to 70 by 15 to 20 μ. The *ascospores* are hyaline, elliptical, unequally two-celled, slightly curved, and measure from 18 to 28 by 4 to 6 μ. They are discharged through an apical pore in the blunt apex of the ascus.

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[1] Class Ascomycetes, Order Phasidiales, Family Phasidiaceae.

Disease Cycle. Strawberry leaves often survive the winter, and diseased leaves may be found in the early spring bearing both the perfect and imperfect stages of the leaf scorch organism. Wolf is of the opinion that the ascospores are more important than the conidia in primary infection, since they are discharged in great numbers during the early spring months when the new leaves are developing. Under suitable moisture conditions the ascospores germinate within 24 hours and infection takes place by direct penetration of the epidermal cells, in contrast to the stomatal infection of *Mycosphaerella fragariae.* Plakidas claims that infection takes place predominantly, if not wholly, through the lower epidermis. The mycelium develops in the mesophyll and grows into the upper epidermal layer where it forms the subcuticular stroma which gives rise to the acervuli. The whole process from infection to the formation of conidia may take place within a period of 2 weeks. Conidial infection is similar in all respects to that from the ascospores. The conidia are produced throughout the growing season, so that repeated infections may occur when weather conditions are favorable. Water is largely responsible for the dissemination of the conidia, although calyx inoculation may be brought about by bees and other pollinating insects. Ordinarily, serious infections occur only during the wet spring months, but in some seasons a renewed infection period is to be noticed on the young runner plants during the autumn months. As stated above, infections are especially serious when they occur on the calyces of the fruit.

The mycelium of the fungus is intercellular, and haustoria, if present, are difficult to find. The fungus does not appear to be highly toxic to the leaf tissues, as is evidenced by the fact that the cells remain alive for a relatively long period after the thorough invasion of the mesophyll tissue. Leaves often show no serious effect even after numerous purple spots are evident on the surface, and the tissue in these spots appears to be alive.

Control. Sanitary measures, such as mowing and burning the leaves after harvest, clean cultivation, and frequent renewal of plantings may be useful in reducing infections. The extent to which such measures should be followed depends upon the best cultural practices in any particular locality. One very useful and harmless practice is to remove all old leaves before setting the plants in the spring. Quick drying of leaves may be secured by keeping down the weed and grass growth, thus reducing chances of infection on the developing plants.

Selection of resistant varieties often solves the problem of the control of leaf scorch. Since commercial varieties change rapidly over a period of years, the grower must depend upon current information in selecting available varieties for resistance. Some standard varieties of the past decade are rated as follows: very susceptible—Klondike, Bellmar, Glen Mary; moderately susceptible—William Belt, Aberdeen, Missionary; resistant—Aroma, Premier, Dunlap, Progressive, Blakemore.

Spraying for control of both leaf spot and leaf scorch has been highly successful in regions where it has been tried and where the two diseases are destructive. In addition to controlling the disease, the Bordeaux mixture in these sprays has appeared to stimulate plant growth. Five or six applications of a 4-8-100 Bordeaux mixture have been advocated. The practicability of spraying depends upon the prevalence of the diseases, which may vary greatly as to geographic location and seasonal conditions.

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Leaf Blight of Strawberry

Leaf blight was called "*Dendrophoma* leaf blight" by Anderson, who first called attention to this leaf spotting as a serious disease of the strawberry. It is now generally called "leaf blight" to distinguish it from "leaf spot" and "leaf scorch."

History and Geographic Distribution. In 1893 Halsted reported a "new strawberry blight" in New Jersey and New York. In 1894 Ellis and Everhardt described a new fungus on leaves of cultivated strawberry and named it *Phoma obscurans*. Very little attention was given to this disease until Anderson reported it in 1920 as causing rather severe leaf blighting in Illinois. He studied the fungus and concluded that it should be placed in the genus *Dendrophoma*. Later the disease was reported from a number of eastern states and from Canada.

Economic Importance. Usually this disease causes little injury until midsummer or later. It is often confused with other leaf spotting diseases or is regarded as some type of insect injury. During the late season it may become very destructive, often killing most of the leaves of the mother plants and seriously blighting those of the new runner plants. This blighting results in the production of weak plants for the crop of the following year.

Symptoms. The disease is most conspicuous on the *leaves*, although at times it appears on the *calyx*. Usually, the spots on a single leaflet are limited to one to five or six. When first observed, the young spots are uniformly reddish purple and almost circular in outline. If they are near one of the main veins, the spots are elliptical. Later three zones may be observed: an outer purple zone which gradually shades off into the normal green of the leaf, a light brown zone about 5 mm in width, and, finally, a dark brown central area 2 to 3 mm in diameter. The white central area characteristic of leaf spot is never present. If the spots occur on a prominent vein, and especially if on the midrib, the typical V-shaped lesion is formed, with the purpling of the tissue extending fanlike to the border of the leaflet (Fig. 83). This area remains alive for some time but finally dies. When the spots are a centimeter or more in diameter, small black dots, the necks of the pycnidia, appear, scattered over the central area. These sometimes elevate the cuticle so that the central area takes on a silvery appearance. In some cases the pycnidia do not appear until a large area of the leaf is dead. New spots continue to appear throughout the summer and autumn.

Hosts. The leaf blight fungus has not been reported on any host other than the strawberry. It is not uncommon on wild strawberries in Illinois.

Causal Organism. The fungus causing leaf blight is *Dendrophoma obscurans* (E. & E.) H. W. Anderson.[1] No perfect stage has been found, although Alexopoulos at one time considered a *Gnomonia* as the perfect form. Later this fungus (*G. fragariae*) was found to be the cause of a stem end rot and leaf blotch disease and, while often associated with *D. obscurans*, was determined to be a separate species. (See page 434.)

The *pycnidia* of *D. obscurans* are deeply buried in the leaf tissue with a distinct neck extending through and above the epidermis. They are from 200 to 300 μ in diameter. During damp weather the spores issue from the pycnidia in long tendrils or in hemispherical masses. The *conidiophores* are branched, hence the name of the fungus. The *spores* are oblong-bacilloid, hyaline, continuous, and biguttulate when mature. They measure 5 to 7 by 1.5 to 2 μ.

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[1] Class Fungi Imperfecti, Order Sphaeropsidales, Family Sphaeroidaceae.

Disease Cycle. Artificial inoculation has proved that it is rather difficult to produce extensive infection on the leaves. This may account for the limited number of spots on the leaflets under most conditions. The mode of penetration is not known. It has been established that the fungus lives over winter in the lesions on the old leaves which remain attached to the plant and that spores from the pycnidia on these are viable in the spring. While the disease does not appear until midsummer, spraying experiments showed some control when applications were made prior to the ripening of the fruit, thus indicating that primary infection may occur fairly early in the season. Maximum infection results later in the season from the conidia washed from the oozing pycnidia. This may occur during any rainy period when there is sufficient moisture to dissolve the exuded spore masses. In the spring, after the removal of the mulch, it is often possible to find live leaves which have survived the winter on which the typical lesions of the disease may be found. Leaves killed late in the previous season also contain numerous active pycnidia.

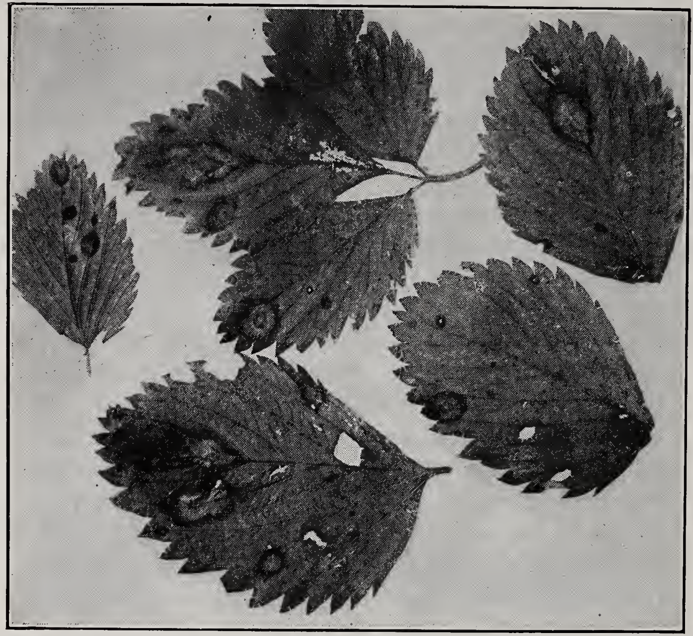

Fig. 83. Strawberry leaf blight. At left, fan-shaped purple area extending to leaf edge.

Varietal differences in susceptibility are marked, but in an examination of a large number of varieties at the Illinois Experiment Station, none were found which could be regarded as highly resistant. Dunlap, Robinson, Red Crop, and Premier are considered especially susceptible.

Control. Little information is available as to effective spraying for leaf blight control. In 1955 Fulton and Cation reported successful control of leaf blight in Michigan by early spring application of Tag 331 (10 per cent phenyl mercury acetate) at a concentration of ½ pint in 100 gal, used at the rate of 150 to 200 gal per acre. The application should be made in early spring, just as the plants are beginning to break dormancy. In some regions spraying may not be warranted.

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Red Stele Root Rot of Strawberry

Various writers have used the names "red core," "red root rot," "brown stele," and "black stele" to describe this disease. In Scotland it was first called the "Lanarkshire strawberry disease" because it was first observed in that region.

History. The first definite reports of this disease are from Scotland in 1927 where Wardlaw attributed the trouble in Lanarkshire to poor cultural conditions followed by the entry of weak root parasites. It was evidently observed in that region shortly after the First World War (1921). Alcock and her coworkers (1929, 1930) reported a *Phytophthora* associated with the disease and deduced that this was the cause of the red core disorder and subsequent death of the plants. They failed to isolate the fungus, but reported that it was probably a new species, although they did not name it.

Bain and Demaree in 1938 reported the successful isolation of the fungus which they did not name although they regarded it as a new species. Alcock and others using diseased roots in sterile soil were able to reproduce the typical symptoms on disease-free plants and were able to find the characteristic oöspores in the stele. Hickman in 1938 isolated the fungus in England and, in 1940, described it as a new species, *Phytophthora fragariae*. After the isolation of the fungus Bain and Demaree conducted an extensive series of experiments on inoculation of plants and made a thorough study of the cultural, morphological, and physiological characters of the fungus. Their results published in 1945 also record attempts to transfer the disease to *Fragaria* species and to unrelated host plants (see Hosts).

In 1930 Anderson observed a disease in eastern Illinois which corresponded closely to the description given by the Scottish workers of the Lanarkshire strawberry disease. In 1935 he reported the disease for the first time in the United States. Specimens sent to Alcock in Scotland confirmed the identity of the causal organism. She was unable to isolate the *Phytophthora*.

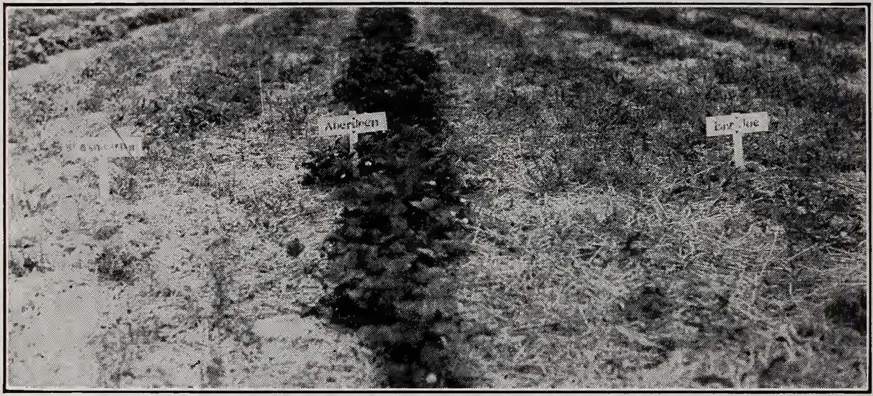

FIG. 84. Discovery of resistance of Aberdeen to red stele. All other varieties in test made scant growth or died. (*Photographed May,* 1935, *Vermilian, Illinois.*)

Pathologists working with this disease soon recognized that the only practical control consisted in securing disease-resistant varieties. Breeding and selection for resistance were first started at the West of Scotland Agricultural College, Glasgow, Scotland. Reid, at the Auchincruive station, began work in 1930 on crossing certain selections which had previously been collected from various sources. This work was continued over a period of ten years, during which a number of highly resistant varieties were secured, several of which were released. Unfortunately, some of the better varieties proved to be susceptible to virus diseases. Work on varietal resistance to red stele was started in Illinois in 1934. Anderson and Colby planted 22 varieties of strawberries in a heavily infested field in Edgar County, Illinois. In 1935 it was found that one variety, Aberdeen, showed no evidence of the disease (Fig. 84). This variety has been utilized extensively in most of the breeding work since the discovery of its resistance. It is reported by Reid and others to be immune. With the discovery of the prevalence of this disease in the eastern states in 1935, the U.S. Department of Agriculture in cooperation with the Maryland Experiment Station began an extensive breeding program to develop desirable resistant varieties.

Geographic Distribution. Red stele has been reported from most of the states where strawberries are grown on a commercial scale except in the extreme South. It has been reported from several counties in England and Scotland and from New Zealand, but not from the European Continent where it was supposed to have originated.

Economic Importance. Strawberries are grown commercially in restricted areas where a number of factors make the industry profitable. If the red stele root rot is introduced into the fields in such regions, it renders the land useless for strawberry growing over a period of 10 or more years. Many growers abandoned growing this crop when most of their available land became infested. Losses are not great for the first 2 or 3 years after the introduction of the disease in a field. It is usually localized in low areas and spreads slowly to well-drained areas. A general distribution of the fungus results in almost complete loss of the crop after the first bearing year. This disease is a threat to any strawberry-growing area.

A very heavy loss is sustained by nurseries producing runner plants. Rigid inspection is required in order to be certain that the disease is not present in the plant bed. The presence of a few diseased plants in a field of several acres makes it necessary to condemn the entire bed, thus causing a loss of thousands of dollars.

Symptoms. The first evidence of the disease on the aboveground parts of the plant may be looked for in the spring of the second year, which is the first year of bearing. The symptoms are first noticed when the plants emerge from the mulch and the leaves begin to extend upward. Those plants which are badly diseased show early evidence of stunting. While the leaves of the healthy plants may be 3 or 4 in. above the mulch, the diseased ones barely extend above the surface. The blades of the leaves of diseased plants do not expand rapidly, but remain small and cupped. Plants which are lightly infected may make an almost normal growth so that they appear, at a distance, to be perfectly healthy. A closer examination, however, will show that the leaf is off color. Leaves of diseased plants take on a lead color, which is evident only when compared with neighboring healthy leaves. The name "wilt" refers to still another symptom of light infection. If the weather has been favorable for the growth of the plant, the leaves and fruit stem may develop normally up to near the time the first fruit is ripening. Then, if a warm spell occurs, the leaves will show a decided droop, especially at midday. This is especially noticeable along the edge of an infected area between the diseased and healthy portion of the bed (Fig. 85).

It is characteristic of this disease to be much worse in one part of the bed than another. Frequently these diseased areas follow a definite pattern, often depending upon the topography of the field. This is directly related to the water-holding capacity of the soil at certain critical times. Rows in a "dead furrow" are often attacked first, or, if there is a draw through the field, the plants in the bottom of the draw are likely to show the first symptoms.

The symptoms so far described are not specific for this disease. In other words, winter injury, or cortical root rot, will give one or more of these symptoms. A certain diagnosis requires an examination of the *roots*. Some evidence of the disease can be found on the roots in the late fall or early winter, but since the tops show no evidence at this time, it is very difficult to locate the diseased plants.

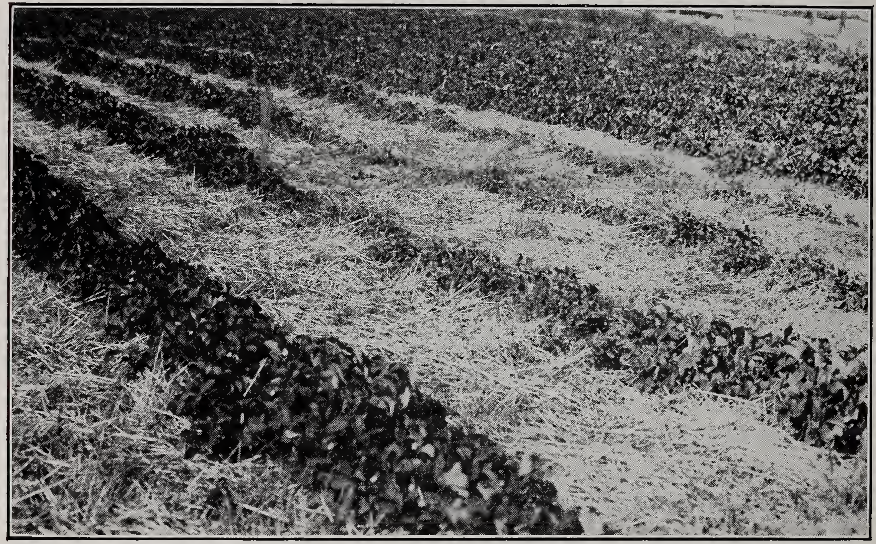

Fig. 85. Red stele showing effect in 1-year-old planting. Photographed in early May at blooming time.

A diseased plant dug in the early spring shows a number of long "rat-tail" roots with a few lateral rootlets. Some of these will appear perfectly healthy, except that they have a grayish cast, as contrasted to the clear white of the normal root. Others will show brown tips where the tissue has started to die. The numerous fibrous roots of the normal plant are lacking. The most characteristic symptom, however, is internal, that is, the red color of the *core* (Fig. 86). By running the thumbnail along the root, the live cortex can be scraped off and the central cylinder exposed. If red stele is the cause of the trouble, the red core will extend well above the dark area at the tip of the root. *To date, no other disease or condition has been found which gives this red core picture.* Later in the spring these diseased roots die completely, and the plant aboveground shows the stunted condition described above. A few roots may survive, or new roots be sent out, so that the plant may show some evidence of renewed growth during the summer. No runners are formed on such plants, and the next year they are dead. Few, if any, berries can be harvested from plants showing any evidence of the disease, since the fruit remains small and often fails to ripen, even though the leaf symptoms are not very pronounced. A bed in which a few plants show the disease during the first year of bearing is almost invariably worthless the second bearing year because of the spread of the disease. Diagnosis of red stele must be made during the period from the start of growth in the spring to about a week or two after harvest. After this time most of the diseased roots have decayed, and no evidence of the disease can be found on such roots as remain alive.

In our varieties the infection stops sharply at the point where the root joins the fleshy stem. In rare cases it may be traced for ¼ in. beyond this point. It has never been found in stolons or other aboveground parts.

Causal Organism. The fungus causing red stele root rot was named *Phytophthora fragariae* by Hickman[1] in 1940. Comparison of this species with other well-known *Phytophthoras* by both Hickman and Bain and Demaree revealed morphological and physiological differences.

Hosts. Bain and Demaree were able to inoculate species of *Fragaria* other than the cultivated strawberry. These were *F. moschata* Duch., *F. virginiana* Duch., *F. vesca* L., and *F. chiloensis*. The fungus has never been found in nature on wild hosts outside the genus *Fragaria*. Species of *Geum* and *Potentilla* growing in infested soil or planted

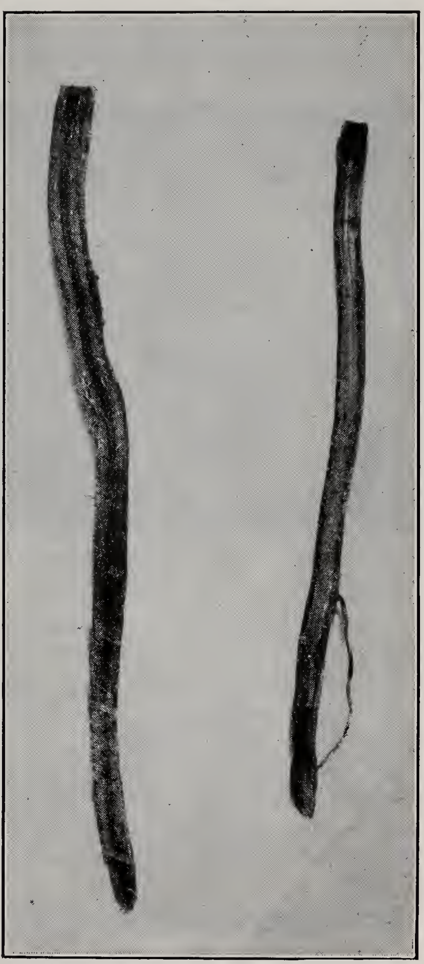

Fig. 86. Strawberry root cut to expose dark core. At right, healthy root with light-colored core.

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[1] Class Phycomycetes, Order Peronosporales, Family Pythiaceae.

in pots containing diseased strawberry plants have never shown evidence of the fungus. Bain and Demaree made inoculations on a number of plants and plant parts with pure cultures in their comparison of this species with other species of *Phytophthora*. In all cases they failed to get infection.

Morphology. The fungus can be cultivated on a limited number of media such as oatmeal agar, steamed bean pods, pea decoctions, and lima bean agar. The rate of growth is very slow as compared to other species of *Phytophthora*. The optimum temperature is between 18 and 22°C. No growth occurs at 30°C, a fact which may explain the absence of the disease during the hot summer months. It grows well at pH ranges from 6.0 to 7.6 and, while it will grow in media with a pH as low as 4.0, no growth occurs at a pH of 8.0 or higher. Sporangia, but not oöspores, are produced in culture.

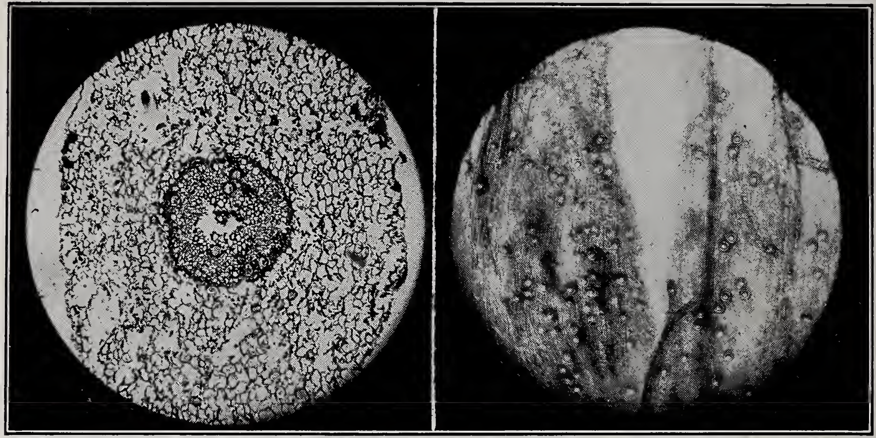
Fig. 87. Right, cross section of strawberry showing oöspores located in the stele. At left, oöspores in macerated stelar tissue.

Sporangia are produced from diseased roots submerged in rain water at low temperatures (15°C). According to Hickman, the sporangia are nonpapillate, inversely pear-shaped or ovoid, averaging 38 by 60 μ. The *conidiophores* may be branched, and proliferation is common. The *zoospores* are ellipsoidal, biciliate, and when rounded out (resting), are 12 μ in diameter. Each sporangium contains 40 to 50 zoospores. Zoospores germinate most abundantly at temperatures from 10 to 14°C.

Sexual Stage. Most of the oögonia and antheridia are produced in the stele. An examination of the roots during the spring months reveals the sexual organs in all stages of development throughout the stelar region (Fig. 87).

The *antheridia* are either paragynous or amphigynous, with the former type predominating. They arise from the base of the oögonium. *Oögonia* are terminal or lateral, variable in shape, averaging about 40 μ in diameter. *Oöspores* are spherical, 25 to 45 μ (average 33 μ) in diameter, surrounded by the wrinkled, dark red oögonial envelope, but with smooth colorless walls, about 3 μ in thickness. Very few observations of oöspore germination have been recorded. Bain and Demaree observed one instance in which the oöspore gave rise to a single long-stalked sporangium which had discharged its contents and had proliferated, forming a second sporangium, also empty at the time of observation. Numerous attempts by various workers to germinate the oöspores evidence the fact that the factors which bring about germination are not yet understood. From consideration of the disease cycle, oöspore germination in nature must be common. It is probable that this occurs during the late fall months, after the disintegration of the diseased roots.

Disease Cycle. The only information concerning the life cycle of the fungus is deduced from the course of the disease as studied under field and greenhouse conditions. The fact that infection may be induced from zoospores indicates that this is probably the chief source of primary infection in the field. This is supported by the fact that the disease spreads rather slowly under field conditions, that it occurs most commonly in wet areas, and that the fungus grows poorly, or not at all, when separated from the living host. The fact that the mycelium is inactivated by a temperature of 30°C (86°F) indicates that the oöspores are the only means by which the fungus can be carried through the hot summer months.

The first evidence of the disease on runner plants is in the late fall when cool weather prevails. Healthy plants planted in the late spring in heavily infested soil but free of diseased plants will grow throughout the summer without evidence of infection. Under Illinois conditions, it has not been possible to find infected roots in new plantings at any time during the fall months. In the eastern United States, fall inspection of strawberry beds is practiced, and it is claimed that the fungus can be found in young roots at this time. Bain and Demaree reported a decrease of oöspores in the roots during the fall and were of the opinion that this resulted from the germination of the oöspores. There is no question but that infection from some source takes place during the late fall and winter months when temperature conditions favor the fungus. It is probable that the sporangia are produced shortly after the germination of the oöspores and that the liberated zoospores swim to the young roots. It is common to find the first evidence of infection on fibrous rootlets or near the tip of the main root.

The fungus grows rapidly through the stelar tissues until it has invaded from half to two-thirds of the root. After this, growth is slowed down, and invasion of the vascular tissues at the point of attachment of the root with the root stalk rarely occurs. In some cases it is possible to follow the fungus into the root-stalk tissues. Hickman claims to have found the fungus in the stolon of diseased plants, but this is certainly unusual. Oögonia are evident shortly after the invasion of the stele. It is evident also that the fungus grows to the surface of the invaded rootlets and produces crops of sporangia from which zoospores are released for further infection. Maximum infection occurs during the early spring months when the runner plants established the previous summer are developing numerous lateral roots and new roots from the crown are pushing down into the lower soil. It is on these long unbranched roots developed in the late fall or early spring that the first evidence of the disease is likely to be observed. The tips of lateral fibrous roots also are invaded, and the fungus grows into the stele of the main root. Many of the lateral roots are killed. If killing is extensive, the new leaves remain dwarfed and there is little extension of the petiole. If only a portion of the root system is invaded, the plant may develop in a normal manner until near the beginning of fruit ripening, when the whole plant wilts. At about harvesttime the invaded portions of the roots blacken and die, although up to that time the cortical portion of the root may be healthy for some distance along the region where the stele has turned red. Invasion of the stele naturally prevents the development of lateral roots, which normally originate in the stele. It is difficult to find the mycelium after this period, and it seems evident that, with the maturing of the oöspores, most fungus growth ceases.

Whether this fungus, like other soil-inhabiting *Phytophthoras*, grows through the soil and invades roots distant from diseased plants has not been established. The fact that it grows poorly on most media and that it is restricted largely to a particular area of the host plant is evidence that it does not invade the soil to any great distance, if at all. Its relation to wet-soil conditions and the slow spread of the disease in the field from an infected area indicate that spread from plant to plant or from diseased to healthy roots of a plant is dependent upon zoospore production and dissemination.

Dissemination. The fungus is frequently introduced into an uninfested soil by planting infected plants. Growers are of the opinion that it can be spread from field to field on the feet of pickers or other workers. There is no good evidence on this point, but it would seem unlikely unless oöspores were present in the surface soil. Well-recorded observations are plentiful of the spread of red stele from washing of soil from an old diseased planting to new beds, and the spread of the disease from one portion of a field to another is often plainly indicated by observing the contour of the field. In the first year all the plants in the path of drainage from this area will show the disease, while the remainder of the field may remain entirely free.

Cultivation, especially in the renovation of a planting, is likely to spread the fungus and cause rather general infection the following year.

The spread of the disease from plant to plant may be from zoospores released into the soil water. The period of activity of zoospores has been determined to be 3 or 4 hours. Thus they are not likely to travel very far unless aided by water movement. Zoospores are discharged and germinate readily at temperatures near freezing and thus may infect during the winter months. Temperatures above 75°F are very unfavorable for zoospore production, and it is probable that no infection from this source occurs during the late spring and summer months.

The appearance of red stele root rot during the past twenty years in widely scattered localities throughout the United States clearly indicates that infected plants were responsible for its wide dissemination. In Illinois where the disease was first recognized, an extensive survey of wild strawberry plants in woodlots and fence rows adjacent to infected fields failed to reveal any evidence of the disease. It has never been found on wild strawberries, although thousands of plants in various parts of the state have been dug and the roots carefully examined.

Physiologic Races. The breeding of strawberry varieties for resistance is complicated by the fact that a number of physiologic races of *Phytophthora fragariae* have been found. In North America at least three such races have been demonstrated, and it is claimed that there are four such in Britain. Two races in the United States seem to be widespread, and attempts are being made to breed varieties resistant to both of these strains.

Control. Rigid inspection of plants in nurseries may serve to reduce the chance of infection, but this method of control cannot be depended upon once the nursery has been found to harbor the disease. Growers who have plenty of suitable locations for growing strawberries can depend on securing a crop for over a 2- or 3-year period by using inspected plants or by growing their own plants in uninfested soil. If the disease appears during this time, the field should not be replanted.

Rotation of crops is of doubtful value. It would seem from experience in Scotland and the United States that the fungus remains in infested soil for a period up to 8 years. In Illinois an infested field was planted to corn and other crops for a period of 4 years, after which it was replanted to strawberries. Plants from this same lot were planted in an adjacent field in which strawberries had never been grown. The infested field showed a severe infection of red stele the following year, while the control field showed none.

Soil moisture is an important factor not only in the spread of the disease but also in its prevention. In some areas of good soil drainage the disease either fails to appear or does no great damage. Sandy soils with a good subsoil drainage are best for preventing loss, provided proper cultural conditions can be maintained.

Use of resistant varieties seems to offer the only certain method of red stele control. Fortunately, a number of varieties have been developed which are highly resistant, if not immune to the disease. On the other hand growers are reluctant to use these varieties because they are either of inferior market quality or because certain standard but susceptible varieties have become so well established that the buyers will not buy the resistant varieties under an unknown name.

Antibiotics were used with success in Connecticut to control red stele. Other soil disinfectants, such as those used in California for the control of *Verticillium* wilt, may give some control, but more experimental work on this method of control is necessary before it can be recommended.

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Cortical Root Rot (Black Root) of Strawberry

The term "cortical root rot" is applied to a root rot condition which primarily affects the cortex in the early stages, although in advanced stages both the vascular system and the cortex are involved in the general decay. This is in contrast to the condition existing in the red stele disease, where the fungus develops in the vascular system, the "core," or stele, while the cortex at first is not killed, although later this tissue gradually becomes discolored and finally dies. The term "black root" was used by several early writers and consequently has priority over the name here used, but it lacks in being specific and descriptive. Cortical root rot may be confused with the natural brown or black condition of the old main roots at the end of the growing season or in the early spring before new roots are formed.

It is best to speak of this disease as the cortical root rot *complex*, since no single organism has been found constantly associated with the disease in any of the regions where it has been extensively studied. In this treatment an attempt will be made to summarize the results of investigations of some of the causative agents and the conditions which are conducive to the development of the disease by these relatively weak parasites.

History and Geographic Distribution. During the past half century complaints by strawberrygrowers have been constantly received by experiment-station workers to the effect that newly planted beds soon deteriorate to the extent that in the second and third year the plants are sickly and "spotty," with greatly reduced yields. This "degeneration" has been ascribed to a number of causes such as virus diseases, winter injury, nutritional conditions, root drowning, insects, nematode injury, etc., but in many cases the rotting of the roots has been the most pronounced symptom observed. Thus plant pathologists became interested in the possible parasitic fungi or bacteria involved. In the United States the general experience of commercial strawberrygrowers was that new beds should not be planted where strawberries had been grown previously, even after a lapse of 3 or 4 years during which other crops had been grown.

In 1917 Fletcher described a "black root rot" which he stated had been prevalent in New York, Michigan, and Massachusetts in 1902 and 1908. Heald in 1920 mentioned the serious dying out of strawberries in Washington and ascribed the trouble to *Rhizoctonia.* In 1922 Smith and Horne described a cortical root rot in California, but did not find any definite organism associated with it. In Michigan Coons published a report in 1924, calling attention to the "black root" of strawberry, and ascribed it to *Rhizoctonia.* Sherbakoff in the same year stated that cortical root rot was common in the southern states and isolated a sterile fungus from many of the specimens but did not identify it. About this time Berkeley in Canada began an investigation of the disease since it appeared to be causing serious losses in the Ontario region.

During the thirties the investigations of the "black root" condition were carried on in both North America and England. These resulted in a series of publications by Strong and Strong in Michigan, Berkeley and Truscott in Canada, and Berkeley and Lauder-Thompson in England. Reports from France and Australia during this period indicated that the root rot condition was general in many parts of the world. In these investigations no one organism was found which could with assurance be held responsible for the root rotting, although a species of *Rhizoctonia* was found by several investigators as being constantly present.

It is held by some that cortical root rot is primarily winter injury to the roots and that fungi associated with it are of minor importance. [See H. E. Thomas, *Phytopathology,* **18**:245–246 (1928)]

Organisms Associated with the Cortical Root Rot Complex. By 1940 the following fungi had been reported as associated with the cortical root rot and capable of causing the breakdown of cortical tissue under certain conditions when the roots were inoculated: *Coniothyrium fuckellii* (*Leptosphaeria coniothyrium*), *Hainisia lythri* (*Pezizella lythri*), *Ramularia* sp. (*Cylindrocarpon radicicola*), *Fusarium orthoceras, Pachybasium candidum,* and *Corticium vagum* (*Rhizoctonia*). In addition, certain phycomycetous mycorrhizal fungi were suspected of being obligative parasites, and several species of *Pythium* were found to be commonly present, some of which were proved to be definitely parasitic. Nematodes have also been mentioned as agents responsible for cortical root rot.

Symptoms. A plantation affected by root rot presents an uneven "patchy" appearance due to dwarfing of the diseased plants and to gaps caused by the complete death of the severely affected plants. It is generally agreed that certain definite symptoms are characteristic of cortical root rot, regardless of the organisms concerned. Under certain environmental conditions some of these organisms are considered much more virulent than others.

A healthy runner plant, when examined in the early spring, will show a cluster of 10 to 20 main roots arising from the base of the crown, with numerous lateral roots and root fibers arising from the lower three-fourths of the main roots. All of these roots and rootlets will be white or light tan in color. Old mother plants will show a cluster of the old roots, brown or black in color with white- or tan-colored adventitious roots arising from the side of the crown above the old root cluster.

The first evidence of infection is the appearance of brown areas on these white or light tan *roots.* The lesions may be elliptical and on one side of the root or extend entirely around the root. These diseased spots may appear in the spring or later in the season on both the main and the lateral roots. Often the small fibrous *rootlets* are killed back to the roots from which they arise. The localized lesions soon extend up and down the roots and into the deeper tissues, so that the entire root may be blackened. The black spongy *cortex* sloughs off if scratched with a fingernail, revealing the stelar tissue which may be almost normal in appearance or slightly browned. Later the vascular tissue also decays, so that the entire root breaks off when bent or pulled, leaving a short stub at the crown.

Shortly after the rather general invasion of the root system, the aboveground parts of the plants show the effect. The leaves are small, often yellowish, and with short petioles. Later, following dry, hot periods, the older leaves may wither or burn around the margins. The fruit stems are usually shortened, and the berries if formed remain small and often wither before ripening. The interior crown tissue becomes brown, dry, and spongy. During the second growing season some new adventitious roots may arise from the area of the crown above the decayed roots, but these are often weak and lacking in lateral root development. These in turn are infected so that by the third year the plant, if alive, is no longer productive.

Economic Importance. It is difficult to arrive at definite figures of losses. When berries are planted in "sick" soil, the loss the first bearing season may not be great, especially if the initial infection is light or occurs late. The crop of the second year may be reduced as much as 50 per cent, and in the third bearing year the crop may not pay for the cost of picking. Thus it is a common practice of growers to plow up beds after two crops are harvested. It is true, however, that agents other than parasitic fungi are often concerned in the general degeneration of the strawberry and that even in the absence of cortical root rot there may be considerable reduction in yield in the second and third years of bearing. The short life of the bed and the necessity of planting on new land are important factors in estimating losses.

Causal Organisms. As stated above, a number of fungi are known to be able to produce cortical root rot under certain conditions. The relation of the extent and severity of the disease to such factors as the cover crops preceding the planting of strawberries was extensively investigated in Ontario by Hildebrand and his associates. Their results, as published in 1941, indicated that the microflora of the soil had a marked effect on the amount and severity of the root rot, although these microorganisms were not in any sense to be regarded as parasites on the strawberry roots. They showed that plants grown in sterilized root rot–"sick" soil increase over four times in aggregate weight over those grown in unsterilized soil; that following crops of soybeans the aggregate weight in the unsterilized sick soil was about 2½ times that of the plants from the untreated soil (control), and that following crops of red clover the aggregate weight was only slightly higher than the control. Following other crops, intermediate results were obtained. Seeking an explanation of this difference, they made a study of the bacterial flora in the rhizosphere of the strawberry roots in the various treatments as contrasted with the bacterial population outside the rhizosphere on the same soils. They concluded that the bacterial balance in the rhizosphere was important. The group of bacteria which brought about a beneficial effect included predominantly carbohydrate fermenting organisms, rather than putrefactive types such as occurred when chopped clover was added to the soil. As stated by West and Hildebrand, "In general, it appears that the biological oxidation of carbohydrate materials in strawberry root rot soil reduces the severity of the disease by bringing about a marked modification of the bacterial and fungal floras of the soil in such a way that potential 'harmful' organisms are greatly reduced and relatively more favorable microbiological balance becomes established." They suggest that turned-under soybeans produce a beneficial effect on root rot–sick soil, while clover or timothy has little effect.

To summarize, cortical root rot is incited by a number of soil-inhabiting fungi of weak parasitic potentialities which increase and become active in invasion under conditions favoring an unbalanced biotic equilibrium.

Control. New plantings should not be made in areas where strawberries have been grown recently. Plants for setting should be examined to see that they are relatively free of cortical root rot. Soil likely to be water-logged or with poor drainage should be avoided.

Not enough is known concerning the effect of cover crops, but red clover should be avoided and soybeans favored.

In California Wilhelm reported fairly good control of cortical root rot (black root) by soil treatment. He made two applications of a Shell product CBP55 ("technical chlorobrompropene") at the rate of 15 gal per acre. *Verticillium* wilt was also controlled. (See page 433.)

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Fruit Rots of Strawberries

While a large number of fungi and bacteria have been isolated from rotting strawberry fruit, the only ones which are responsible for constant and extensive losses are *Botrytis cinerea* causing "gray mold" rot and *Rhizopus nigricans* causing "leak." The first is the most important since it is more likely to cause extensive damage in the field, while the *Rhizopus* rot is more often a market disease problem.

Three other rots, "*Rhizoctonia* brown rot," "leather rot," and "tan brown rot" (*Pezizella* rot) have been described as common in some areas, but in general are of minor importance.

Gray Mold of Strawberry

This disease has been referred to as "dry rot" and "brown rot," but the gray fuzzy coating produced by the fruiting of the fungus on the surface of the diseased tissue is so characteristic that the name "gray mold" is justified, especially since several other rots produce a brown coloration on the fruit. It is true, however, that the gray fruiting of the fungus is often absent in the early stages of the disease and appears only under moist conditions.

History and Distribution. The fungus causing this rot was one of the first described by mycologists. It is impossible to trace the history of the disease on strawberries, since in early horticultural literature there was no distinction made between the various types of rot on this crop. Since the fungus occurs on a large number of hosts and is common as a saprophyte on decaying vegetable matter and since it has a world-wide range, it is probable that it has always been associated with the cultivated and wild strawberries as one of the main agents of rot. As a strawberry rot it has been reported from most countries where this crop is grown. The disease is especially prevalent in regions where rainfall or dews occur frequently during the blossoming and fruiting period.

Symptoms. The disease, while regarded as a fruit rot, often starts early in the season as a *blossom blight.* In the cluster of blossoms on the main fruit stalk, one or more will show a blasted condition, usually with the disease extending part way down the pedicel. Later, as the berries enlarge, the disease may be observed on the tips of the calyx lobes, often confined to one or two of the lobes. Infection on the *berries* may appear at any point. Often the first rot on half-grown fruit appears at the base of the fruit and originates from the infected calyx. In other cases it may appear on the side or tip, especially where the berry is in contact with the soil or other damp surface. Frequently, following early blossom or fruit infection, the disease progresses downward through the pedicel to the main fruit stem, and finally most of the fruit cluster is involved.

On the fruit the rot shows as a light brown area, somewhat soft at first but later drying out as the whole berry becomes involved. When damp weather prevails the infected blossoms and fruit become coated with the fine gray fuzzy fruiting stalks and spores of the fungus (Fig. 88). Ripening fruit shrivels into a dry leathery mummy. However, in packed, mature fruit the tissues are likely to remain soft, somewhat watery, and easily mashed, although rarely "leaky" as in the case of *Rhizopus* rot.

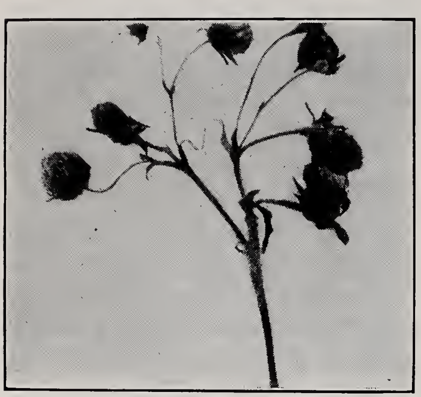

FIG. 88. Gray mold on strawberry fruit, fruit stems, and blossoms. Note fuzzy covering of rotted fruit.

Under damp conditions small black bodies, the sclerotia, may be formed along the withered fruit stems and occasionally on the rotted berries. These sclerotia may be formed also at the base of the fruit stem in the "scales" of the crown when the fungus grows down the main stem.

Economic Importance. Disastrous losses result when excessive rainfall occurs during the period from blossoming until final harvest. For example, in southern Illinois in 1946 there were 22 days out of 34 when rain fell during this period. In that year a number of growers in this region estimated a direct loss by fruit rotting of from 50 to 70 per cent of their crop. While this is exceptional, there are few years when some loss does not occur. It was not realized until recently that considerable loss in production may occur, with little evidence of the disease on the harvested berries. This fact was brought out when effective sprays were applied for the control of gray mold. In one such experiment the yield from the sprayed area was almost double that from the unsprayed part of the field. In this case not many rotted berries were found in the harvested fruit from the check plot, so that it was evident that blossom blight and early-fruit infection were reducing the yield. The increased yield might, of course, be due in part to the control of other diseases, but these were of minor importance in this experiment.

The loss in transit and market may be extensive if care is not taken in grading the fruit in the field, and prompt shipment under refrigeration is essential.

Causal Organism and Disease Cycle. The gray mold rot is caused by *Botrytis cinerea* Fr.[1] At one time it was considered by some to be the imperfect stage of *Sclerotinia libertiana*, a common parasite on lettuce and grape. Others considered that this relationship had not been definitely established and regarded it as possibly a *Sclerotinia*, in which the ability to produce the sexual stage had been lost. In 1939 Groves and Drayton succeeded in producing apothecia of *Botrytis cinerea* in culture, but it is generally recognized that in nature the fungus produces no sexual stage, or if it is produced, it plays no part in the disease cycle. It is probable that biologic strains of this species occur, but this has not been investigated so far as the fungus commonly found on strawberry is concerned.

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[1] Class Fungi Imperfecti, Order Moniliales, Family Moniliaceae.

The *sporophores* (spore stalks) arise thickly but singly from the diseased organ and extend from 3 to 5 mm above the surface. They are dark in color except at the tip. The branches arising from this tip are short and crowded, so that the spores are borne in a grapelike cluster. The spores are uncolored; the gray to grayish black appearance is due to the dark color of the sporophores beneath the spore masses. After a crop of spores is produced and shed, the branches of the main stalk die back, the conidiophore extends upward, and another series of branches and spores is produced. The *spores* (conidia) are single-celled, ovate, hyaline, and measure 8 to 11 by 11 to 15 μ. The spores are easily detached, and light currents of air or jarring will give rise to a cloud of spores. The black *sclerotia*, about the size of a wheat grain, are formed on any diseased organ, but usually are few and scattered. They are more commonly found at the base of the rotted fruit stalk or on the old rotted berries in contact with the mulch or soil. These sclerotia are important in the life cycle, since they are resistant to all climatic conditions. In the spring they give rise to a crop of spores which are carried by currents of air to the developing blossoms or fruit, where in turn another crop of spores is produced, thus spreading the disease. The source of the spores for initial infection may not be from the strawberry, since the fungus occurs and fruits on any damp, decaying vegetation where it may form sclerotia for winter survival.

disease
cycle.

In the presence of moisture the conidia germinate in a few hours and the germ tube is able to penetrate uninjured tissue. In the blossom it is likely to start development on the stigmatic surface of the pistil. Another favored court is at the base of the berry where the subtending calyx prevents rapid drying.

After gaining entrance the mycelium dissolves the middle lamella of the cells, thus at first giving rise to a rather soft rot. Later the hyphae become intracellular through penetration of the cell walls. As the mycelium grows through the fruit tissues, it apparently takes up enough of the watery material to dry out the tissues so that in the end the rotted areas are rather firm or even leathery.

Environmental conditions are of great importance in the development of the disease on the strawberry. If the period from blossoming to early harvest is relatively dry, infections are so few that a minimum loss occurs. A rainy period during bloom followed by either light rains or dews as the fruit matures is conducive to the rapid spread of the disease. During the first picking the fungus spores on a few rotting berries may be spread to neighboring clusters handled by the pickers, or these spores may lodge on the picked fruit, and the rot may appear in transit or in the market. Often the disease appears only on fruit clusters near the center of the matted rows, where slow drying of the plants is to be expected.

Temperature is of little importance under field conditions except as it influences moisture relations, but it is of great importance in the development of the fruit rot in the package fruit.

Control. Sanitation and cultural practices will aid in control. Where the variety grown produces numerous runner plants resulting in matted rows, a judicious spacing by thinning should be practiced. This permits better air circulation and consequent rapid drying. Where heavy winter mulching is practiced, only enough mulch should be left in the rows to prevent the fruit coming in contact with the soil. Pickers and graders should be trained to watch for fruit with withered calyces and discard such berries even if no rot is apparent on the fruit.

Spraying strawberries for disease control has not been generally practiced in the United States in the past, but recently has become an established practice in many of the main strawberry-growing areas. For control of gray mold, applications should be made just about the time the first blossoms are opening, followed by two applications spaced so that the second application is made about the time the first fruit starts to color. Captan (50 per cent) used at the rate of 6 lb per acre per application successfully controlled the disease under Illinois conditions. Spray nozzles in the boom should be arranged so as to drive the spray into the row from above and from the sides.

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Rhizopus Rot of Strawberry

This disease is commonly called "leak," since the juice from the diseased mushy berries stains the boxes and leaks out of the bottom of the containers. It is primarily a market disease, although it is not unusual to find it rotting ripe berries in the field. In shipped berries it often accounts for over 50 per cent of all the rots, with gray mold the next most abundant. Since this is primarily a ripe rot, the field loss is not great, provided the berries are picked at the proper stage of maturity. The disease occurs only on the berries, although the fungus will develop and fruit on any moist surface.

Symptoms. *Rhizopus* rot is characterized by the very soft watery tissue at the surface of the fruit and the ready exudation of the juice on slight pressure. This fruit may retain almost its normal color for a while after the invasion of the fungus, but soon changes to a light brown. In the packaged fruit the berries may become covered with a white fluffy growth which extends across the intervening space between the berries and even alongside of the container (Fig. 89). Black fruiting "heads" may be seen scattered over the surface of this growth, provided the fungus has been established for some time. These fruiting structures are more scattered and darker than in the gray mold rot.

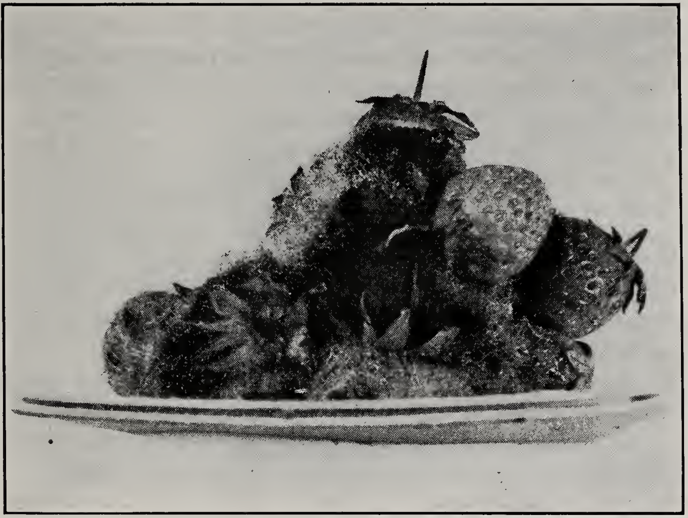

FIG. 89. *Rhizopus* rot of strawberry, cluster of rotted berries bound together by mycelium with dark fruiting "heads" of fungus covering mass.

Causal Organism. The fungus causing the rot is the common "bread mold" *Rhizopus nigricans* Erh.[1] This fungus is almost omnipresent since it grows and fruits on all types of vegetable matter and causes rot of a large number of fruits and vegetables. It is a Phycomycete of the order Mucorales in which zygospores are produced in the sexual stage. The *mycelium* is nonseptate, and the individual hyphae are so large as to be easily visible to the naked eye. Two types of spores

[1] Class Phycomycetes, Order Mucorales, Family Mucoraceae.

are produced: *asexual* in sporangia and sexual *zygospores* formed by the union of two like gametes. The asexual stage is the only one of importance in the dissemination of the fungus. When the asexual spores germinate they produce a loose web of mycelium, which under moist conditions extends in a white fluffy mass above the substratum. The rapid and extensive growth in closed containers has given it the common name of "whiskers." Some of these erect hyphae bend over, and when the tip comes in contact with a solid surface, it enlarges and gives rise to several erect *sporangiophores*. Next to the substratum of this enlargement are produced a number of rootlike anchoring hyphae. At the same time a single strand extends horizontally for a short distance and again forms a cluster of sporangiophores and root hyphae. This fruiting strand is called a *stolon* and produces an effect in miniature much like that of a series of plants on the runner of the strawberry. The tip of the erect sporangiophore enlarges into a globular head into which the rich granular protoplasm flows. This head is then "plugged" by the formation of an arched wall which swells out into a smaller globular structure, the *columella*, within the enlarged *sporangium*. From the granular mass of protoplasm thus entrapped, a large number of segments are cut out which soon round out, finally forming a mass of black, powdery *spores*. These clustered "heads," scattered over the surface of the mold, give the black appearance so often observed on rotted fruit. The outer wall of the sporangium is easily ruptured, so that the powdery mass of spores is released to float about in any air current or drop to the surface of the substratum. The spores are subglobular and measure from 6 to 17 μ in diameter.

Rhizopus nigricans is a heterothallic species, therefore the sexual spores are produced only when unlike strains come in contact. Since the sexual cells (gametes) are alike, these strains are designated as plus (+) and minus (−) strains. The zygospore when mature has a dark thick wall studded with hemispherical warts. It measures from 160 to 220 μ.

Disease Cycle. *R. nigricans* is a wound-invading pathogen and for this reason is more likely to be found on ripe fruit which is more subject to bruising and wounding than green fruit. Often no indication of wounds can be detected at the point of infection. Apparently sound peaches, for example, are often found to be infected when the whiskers from neighboring fruit makes contact with them.

The spore on germination produces a germ tube which, if it comes in contact with the wounded surface of the fruit, penetrates between the cells, dissolving the middle lamella as it progresses. If the spore germinates on an unwounded surface, the germ tube branches and forms a weft of hyphae, some of which may find a point of entry and start the rotting process. In the strawberry fruit, according to Stevens, only the outer six or eight cell layers of the pulp are invaded, while the interior remains relatively free of the mycelium. The growth of the fungus between the cells results in a collapse of the adjoining cells, with an apparent accumulation of juice in the intercellular spaces, thus giving rise to the "leak" condition. The fungus apparently does not penetrate the cells, at least until they are collapsed.

In the packaged fruit much of the spread of the disease is from the hyphae growing from the surface of the previously rotted berries rather than from spores which may be formed in the package. In the field, however, the infections are likely to be from the omnipresent spores. These spores are highly resistant to desiccation and other climatic factors. The fungus may pass the winter in the mycelial stage in vegetable matter.

Relation to Temperature. The fungus grows and fruits very rapidly at high temperatures. At 85°F. *Rhizopus* will produce mature sporangia in 36 hours, while at 50°F it takes 3 weeks. This emphasizes the fact that quick cooling of the harvested berries is essential.

Control. Since the spores are omnipresent, any wound, however slight, is likely to be a court of infection. For this reason bruised or injured fruit should be discarded by the pickers and graders. Overripe fruit should not be packed with firm ripe fruit.

Precooling the fruit before shipment is very essential. If the temperature can be reduced within 4 to 5 hours to 50°F and kept at this temperature during shipment, very little rot will develop, although the berries may be infected when packed. Fortunately, under present marketing conditions practically all quality berries are kept at low temperatures in the market.

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Minor Diseases of the Strawberry

Stem-end Rot and Leaf Blotch. This comparatively unimportant disease was called "leaf blotch" by Fall in Canada and by Wormald in England since they found the fungus on the leaves. In Michigan where it caused a fruit rot it was called "stem-end rot" by Alexopoulos and Cation.

In 1941 Wormald and Montgomery reported a leaf disease of strawberries, the causal organism of which they identified in 1944 as *Zythia fragariae.* This fungus was also found on the fruit. In 1918, Klebahn described a fungus on strawberry foliage which he named *Gnomonia fragariae.* This later proved to be the perfect stage of *Z. fragariae.* The

Arnauds in France in 1931 reported the isolation of this same fungus from strawberry fruit. Alexopoulos and Cation in 1948 reported that a *Gnomonia,* which they at that time considered the perfect stage of *Dendrophoma obscurans,* the leaf blight fungus, was present on the calyces of strawberries and was causing a stem-end rot of the fruit. Later they concluded that the imperfect stage of this fungus was *Z. fragariae* and not *D. obscurans,* thus agreeing with Wormald, Klebahn, and the Arnauds. In 1951 Fall reported the disease on strawberry leaves in Canada. She stated that it was often associated with *D. obscurans,* as was found by other investigators. She confirmed the fact that the imperfect stage was *Z. fragariae.*

Symptoms. There is some question as to whether the fungus is parasitic on the leaves. On the fruit, however, it withers the calyx and progresses into the base of the fruit where it causes a light brown, rather firm rot.

Causal Organism. The perfect stage of this fungus (*Gnomonia fragariae* Kleb.)[1] is rarely found in nature but is readily produced in culture on some media. The perithecium is globose, with a long neck, which gives the whole structure a flasklike appearance. When found on the plant it is embedded in the host tissue, with the long neck extending above the surface. The base measures 450 to 600 μ in diameter, the neck being 500 to 800 μ in length. The slightly curved ascospores are two-celled when mature and measure 10 to 12 by 2 to 3 μ. Unlike most Ascomycetes the ascospores are not forcibly discharged but ooze out of the ostiole after being released by the deliquescence of the asci.

On the whole this disease seems to be erratic in its appearance and is considered of minor importance. The limited information indicates that it may at least cause serious fruit rotting under favorable conditions. Also, *Zythia fragariae* has been mentioned as one of the common species isolated in investigations of the cortical root rot complex.

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Verticillium Wilt of Strawberries. Damage to strawberry plantations due to *Verticillium* wilt has focused attention on this disease during recent years. This increase in interest may be the result of a better understanding of the disease which in the past was confused with the black root rot complex. Both in the United States and England the importance of the disease has been emphasized in recent years. In England both *V. albo-atrum* and *V. dahliae* are regarded as causal agents, while in North America *V. albo-atrum* is most often mentioned.

A full discussion of the *Verticillium* wilt problem is given under Bramble Diseases (page 318).

The *symptoms* of *Verticillium* wilt on strawberries are such that it cannot be clearly differentiated from other root rot diseases. Wilting of scattered, individual plants, with slight browning of the crown tissue, is one of the characteristic symptoms, but this may occur with other diseases or from winter injury.

This disease is of minor importance in most regions where proper care is exercised in planting strawberries in soil not heavily infested with the fungus. It is especially important to avoid planting new beds following crops of solonaceous plants such as potatoes, tomatoes, and eggplants.

Wilhelm, in California, in reporting on this disease, stated that as high as 65 per cent of the strawberry plants were killed by this fungus when planted on infested soil. In England it was demonstrated that runner plants from a plantation showing a high incidence of the wilt when planted in noninfested soil showed no evidence of the disease, while if planted in infested soil, most of the plants were killed during the first season.

Control. Wilhelm was able to reduce the loss of plants from over 65 per cent to 5 per cent by two applications of CBP-55 at the rate of 15 gal per acre, making two applications in the fall to the infested soil. This method of control also gave excellent control of black root rot (cortical).

There is a marked range in pathogenicity of strains of *Verticillium albo-atrum* as well as in the susceptibility of strawberry varieties to the disease. Thus in one region a variety may show high resistance, while severe damage may occur to this variety in another region.

The organism causing this disease is a common inhabitant of the soil. If the strain parasitic to strawberries is present in the soil, there is little hope of effective control. In general it is not advisable to plant strawberries following solonaceous crops which have shown evidence of the disease. Investigations now under way on strawberry varieties resistant to *Verticillium* wilt may give some indication of varieties which may be grown safely in some regions.

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Leathery Rot of Strawberries. This disease is caused by a soil-inhabiting *Phytophthora* (*P. cactorum*), and like the *Rhizoctonia* rot usually occurs only where the berries come in contact with the soil. The fungus attacks berries in the field in all stages of growth. On green fruit the surface of the rotted area turns brown and appears water-soaked. There is no line of demarcation between the infected and healthy tissue when the fruit is sectioned. The pulp tissue is not softened or watery but remains rather firm. On ripe fruit the rot finally results in a mushy condition of the fruit. The vascular bundles in the green fruit are discolored, a characteristic symptom that does not occur with other rots. The fruit has a bitter taste. For details in regard to the fungus *Phytophthora cactorum* see *Phytophthora* Collar Rot of apples (page 119).

The disease is of little economic importance except in areas where the beds are not mulched. [D. H. Rose, Leather rot of strawberries. *J. Agr. Research*, **28**:357–375 (1924).]

Rhizoctonia Brown Rot of Strawberries. In 1924 Dodge and Stevens reported on a fruit rot of strawberry commonly observed in the market on fruit shipped from Florida. Observations in the field revealed the fact that this rot was rather common, and many berries were culled out during picking and packing.

Affected berries are generally one-sided and show a hard brown rot quite different from other brown rot diseases. Decay starts on the underside of the berry where it comes in contact with the soil, and often particles of soil and sand cling to the rotted area when the fruit is harvested. There is a definite line of demarcation at the margin of the rot, indicating a slow and limited growth of the fungus in the fruit pulp. The rot does not extend beyond this limited area even after the fruit is harvested and packed. The brown strands of the mycelium may be observed with a hand lens on the surface of the lesion and in the dried-out interior pulp.

This rot has been reported mostly from southern United States, but since the fungus causing it is one of the commonest soil-inhabiting fungi, it is probably present to a limited extent wherever strawberries are grown. It is not likely to be of importance where mulch is used and the berries do not come in contact with the soil.

The fungus concerned is a *Rhizoctonia*, probably the common *Rhizoctonia solani* (*Corticium vagum*). This fungus is very commonly found in strawberry roots suffering from cortical root rot (page 420). The disease is of minor importance. A layer of loose mulch preventing contact of the berries with the soil will control the disease. [B. O. Dodge, and N. E. Stevens, The *Rhizoctonia* brown rot and other fruit rots of strawberries. *J. Agr. Research*, **28**:643–648 (1924).]

Powdery Mildew of Strawberry. Occasionally a powdery or surface mildew causes some damage to strawberry plants, but on the whole the disease is of minor importance to the industry. It was first reported in England in 1854 and in the United States in 1886. Outbreaks have been reported at various times since that date in the northeastern and Pacific Coast states.

The fungus *Sphaerotheca macularis* (Wallr. ex Fries) W. B. Cooke occurs on a wide range of hosts including many common weeds, but it is probable that a biologic form of this species is responsible for the strawberry mildew, since in the presence of abundant infections on the weeds the disease is not present on the neighboring strawberry plants. This fungus causes one of the most serious diseases of the hop.

The edges of affected leaves curl upward exposing the undersurface. The powdery frosty growth of the fungus appears over the lower surface, and the fruit stems and berries may become infected. The fruit often fails to color at maturity.

S. macularis has the usual characteristics of the Erysiphaceae. The powdery appearance is caused by the production of chains of conidia on simple conidiophores. The perithecia are usually abundant on the lower leaf surface as small black dots. These have a few long, colorless, unbranched appendages. The perithecium contains a single ascus with eight large oval ascospores about 20 μ long.

The perithecia survive the winter and the liberated ascospores give rise to new infections in the spring.

Darrow and others in 1954 reported on the relative resistance of strawberry varieties to powdery mildew. They observed that mildew was more severe on the plants in "screenhouses" than in the field. Of 41 varieties 11 were free of mildew and 8 others very resistant. Three varieties (Ambato, Armore, and Royal Sovereign) were killed. The remaining 19 showed slight to considerable leaf rolling and some necrosis.

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Virus Diseases of the Strawberry

Strawberries until recently were considered to be relatively free of virus diseases, although growers realized that favored varieties had a tendency to "run out" after years of cultivation. One reason for the failure to detect these diseases was that many of the cultivated varieties show little effect when virus-infected. Attention was first called to these virus diseases when a very susceptible variety such as the Marshall was grown in a locality favorable for the introduction and spread of the disease. The rapid deterioration of Marshall and related varieties in California and of Royal Sovereign in England excited the interest of plant pathologists and initiated an extensive investigation of strawberry viruses. As in the case of other crops, it was soon realized that several viruses might be involved and that entities within the main virus groups might give entirely different symptoms or no symptoms at all when transmitted to certain strawberry varieties. It was only when satisfactory standard indicator plants were found that rapid progress was made in sorting out the entities involved. Much more work is needed before the picture becomes clear. It is now known that certain viruses are present and widespread in practically all of the commercial varieties and also in wild species of *Fragaria*. It is comforting to know, however, that with few exceptions these varieties continue to be productive and show little evidence of disease. The use of virus-free plants of these varieties in some cases has demonstrated increased vigor and the production of 3 to 5 times as many runner plants even when no symptoms are evident on the virus-infected plants used for comparison. The use of virus-free plants is justified also because of the danger of introducing the virus into new varieties where it might prove destructive.

Geographic Distribution. It is probable that strawberry virus diseases are present wherever strawberries are grown extensively and where vectors are present. They have been reported from Australia, the British Isles, Canada, Continental Europe, Guatemala, New Zealand, South Africa, and the United States. In a limited number of tests virus-infected wild species have been found in both eastern and western United States.

History. Lack of knowledge concerning virus diseases and the obscure symptoms on strawberry account for the fact that we know little concerning the origin of these diseases. The fact that when first noticed they were to be found in widely scattered areas and were so generally distributed in all varieties indicates that they must have been present years previous to their recognition.

In California, Horne, in 1922, was the first to call attention to and describe a disease which was causing trouble on the popular Marshall variety. This disease was first reported from the Watsonville district in

1915. Plakidas in 1927 published the results of an extensive investigation of the disease, which he named xanthosis, or yellows, and proved that it was of virus origin. He demonstrated that it could be transmitted by a commonly occurring aphid and that certain varieties of the Marshall group were especially susceptible to the disease. He found the disease widely distributed in certain areas of California, Oregon, and Washington. About this same period a diseased condition of the strawberries, especially the Royal Sovereign varieties, was under investigation in England. Various factors were suggested as possible causes for the "degeneration" of the strawberry in this country. Among others, Turner in 1923 and Beaumont and Hodson suggested that a virus might be involved, but gave no conclusive proof. Harris of the East Malling Experiment Station in Kent began an extensive series of experiments in which he attempted not only to prove the virus nature of certain of these "degeneration" conditions, but also to sort out some of the entities involved. These investigations started about 1930 and continued over the next twenty years both in England and America and have contributed to a much better understanding of the strawberry virus complex and the development of methods for accurate determination of the entities involved.

In eastern United States it was generally assumed that the standard commercial varieties were virus-free or so tolerant that no injury resulted. About 1945 Demaree, working on small fruit diseases in the U.S. Department of Agriculture, started a series of investigations to determine the extent and nature of the virus diseases in eastern United States. In 1946 he reported that, using the Marshall as an indicator plant, he was able to demonstrate the presence of a yellows-type virus in eastern-grown berries by the runner-graft method of transmission. These investigations were continued in the U.S. Department of Agriculture at Beltsville and in the Northwest over the next eight years. In England, Harris and Prentice also continued their work in an effort to determine what components were involved in the yellow edge and crinkle disease complex. A suitable indicator plant, *Fragaria vesca*, was found, and a clone of this known as the "East Malling clone" became the standard indicator for testing. Rapid progress was then possible not only in determining the type of virus present, but also in securing virus-free plants of standard commercial varieties which could be propagated and distributed to growers by nurserymen.

Plakidas in his investigations in California had found that an aphid, *Capitophorus fragaefolii*, could readily transmit yellows, and this insect was used extensively in his transmission studies.

Miller, working in Oregon, reported in 1951 results of a study on the factors involved in the transmission of the virus by this insect. Frazier in 1951 reported that he was able to get transmission by five other species of aphids, but these were less efficient than *C. fragaefolii*. In the east Demaree and Marcus in 1951 stated that *C. fragaefolii* was rarely encountered, but found *C. minor* and an unnamed species of this genus fairly common, and they were able to transmit the eastern virus types by means of these species.

Harris in 1932 developed a method of stolon grafting which has since become standard in transmission studies, especially in the greenhouse where detailed work on individual plants is undertaken.

Classification of Strawberry Viruses. From the inception of investigations of strawberry virus diseases to the present time, puzzling anomalies in symptom expression led research workers to believe that more than one virus was involved. The separation of these entities based on the use of indicator plants is still incomplete. It is generally agreed, however, that they can be classified in two main groups: (1) yellows and (2) crinkle. In addition, two or three minor virus diseases have been described.

Yellows Group. This is the disease which Plakidas investigated in California and which he called xanthosis. This is considered distinct from the "Blakemore yellows" or "June yellows." (See page 443.) In this group is also included yellow edge, the term used by Harris to describe the Royal Sovereign disease in England. Yellows is considered the most serious and destructive virus disease of the strawberry. Attempts to separate the entities of this group have led to some divergence of opinion.

SYMPTOMS. On all but some symptomless carriers general stunting of growth can be observed when compared with virus-free plants. Upward cupping of leaves, a yellowing of leaf margins, and dwarfing of petioles and blades are common symptoms on those cultivated varieties less tolerant of the disease, such as the Marshall group. The plants do not die, but once infected are permanently stunted and unproductive. Symptoms are often masked at high temperatures.

Two components in the yellows disease have been demonstrated as expressed in indicator plants of *Fragaria vesca*. One has a decided dwarfing effect on this host, producing small, dark green, wrinkled, yellow-spotted or -streaked leaves. The leaflets are small and irregular in shape. Stolons if formed at all are dwarfed. The second component causes moderate stunting and a mild uniform yellowing of the indicator plant. These components and possibly others are involved in the disease as it appears on cultivated varieties in western United States. Demaree and Marcus found that in general no discernible symptoms could be seen on most cultivated varieties but that when *F. vesca* was stolon-grafted to these varieties, definite symptoms appeared in many cases on the indicator plants. These symptoms were of two types which they called Types 1 and 2. Type 1 gave identical reaction to the first component virus. This is believed to be identical with the virus or viruses causing yellow edge in the British Isles and the yellows and crinkle in the West. Symptom expression is found to vary greatly with this virus, which may indicate that still other entities are involved. Type 2 gives very uniform symptoms. On *F. vesca* the leaves are quite small and yellowish green. The leaflets are smooth, normal in shape, and uniform in size, as contrasted with the puckered irregular-shaped and spotted leaflets of Type 1. "Young runner plants are spreading, the older leaves being horizontal and close to the soil, petioles are shorter than normal . . . Later the petioles assume an upright position, and bud proliferations gradually develop from the stem, resulting in multicrowned plants."

When these two types were simultaneously transmitted to a single *F. vesca* indicator plant, marked dwarfing resulted, with many adventitious crowns and extremely small and spotted leaves.

Recent investigations in England which show a close relationship between crinkle and yellow edge point to the fact that crinkle may be one of the components of the yellows group.

Crinkle Group. Zeller and Vaughan in 1932 first called attention to a disease in the Pacific northwest which they considered different from xanthosis as described by Plakidas and to which they gave the name crinkle. They suggested that it was probably of virus origin but did not prove this by transmission from diseased to healthy plants. This experimental proof was furnished by Vaughan in the following year by using viruliferous aphids (*Capitophorus fragaefolii*). According to these authors the most characteristic symptoms are the crinkled condition and chlorotic character of the leaves. The chlorotic areas are localized, starting as pin-point spots on the developing leaves and enlarging as the leaf unfolds. These spots apparently restrict the growth of the tissue and account for the later crinkled appearance of the leaf. The margins of the leaves are irregular, with some of the normal dentations becoming unevenly crenated.

In England, Harris, in 1936, described the results of his experiment from which he concluded that there were two types of crinkle: mild (widespread) and severe. The latter he stated was probably the result of the presence of an additional virus. Plants having both components showed yellow edge symptoms later.

In further investigations by Harris and Prentice in England on the resolution of strawberry virus complexes, they were able to separate three components of the complex by the use of *C. fragaefolii* as the transmitting agent. These were called viruses 1, 2, and 3. The separation of the components was based on the "infection-feeding period" and the "transfer-feeding period." It was found that the minimum feeding period required for transmission of virus 1 was 1 hour, that of virus 2, 24 hours, and of virus 3, 6 days. Virus 1 persisted in the vector for only 3 hours, while the other two persisted for several days. The symptom expression of each of these and of combinations of them was further investigated by stolon grafting.

In 1952 Prentice described two more viruses in the strawberry virus complexes. He gave common names to these five components to correspond to the numbers under which they had been previously described. These names together with the summary of symptoms in the indicator plants *Fragaria vesca* and Royal Sovereign are presented below [the combinations are from an earlier (1949) report].

TABLE 3. VECTOR RELATIONSHIPS OF STRAWBERRY VIRUSES (PRENTICE 52)

Number and name	Acquisition-threshold period	Latent period	Persistence
1. Mottle	About 1 hr	None	Short, 1 hr
2. Mild yellow edge	About 24 hr	None	Long
3. Crinkle	About 24 hr	About 12–16 days	Long
4. Vein chlorosis	....	No vector	
5. Leaf curl	1 hr	None determined	Short

TABLE 4. SYMPTOMS PRODUCED BY STRAWBERRY VIRUSES

Number and name	*F. vesca*	Royal Sovereign
1. Mottle	Chlorotic spotting; leaf distortion	Faint chlorotic spotting
2. Mild yellow edge	Slight chlorotic spotting; slight chlorosis; leaf cupping	Slight chlorosis (mainly leaf margin)
3. Crinkle	Severe chlorotic spotting; leaf distortion; vein necrosis	Chlorotic and necrotic spotting
4. Vein chlorosis	Faint chlorotic spotting	Vein chlorosis and necrosis
5. Leaf curl	Leaf curling; vein and leaf necrosis	Leaf curling; vein necrosis
1 + 2	Chlorotic spotting; severe stunting; sometimes death	Severe yellow edge; stunting
1 + 3	....	Mild form of severe crinkle
2 + 3	....	Severe yellow edge
1 + 2 + 3	....	Very severe yellow edge

Thus one can postulate that the wide variation of symptoms described by a number of investigators may be due to (1) the components involved, (2) the relation of the vector as to feeding time and retention of virus, (3) variety involved, (4) climatic conditions. It is doubtful, therefore, that the separation of the strawberry virus diseases into the two groups, yellows and crinkle, is justified.

Transmission of Viruses. All the components of the strawberry viruses have been transmitted experimentally by aphids. Of the aphids involved, *Capitophorus fragaefolii* is regarded as most effective. *C. minor* and an unnamed species of *Capitophorus*, more commonly found in eastern United States, have proved capable of transmitting the virus. Mechanical inoculation has not been successful. Natural transmission is undoubtedly brought about by the various aphid species which are commonly found feeding on strawberries. The viruses are not transmitted through the seed. Effective transmission is attained by tissue union.

Miller found an increase in successful transmissions when the vectors were subjected to a preacquisition fast period of 3 to 20 hours and also showed that the fasted aphids could acquire the virus with a 30-minute feeding period on the infected plant.

Control. The most satisfactory control consists in the use of virus-free plants. Fortunately, at present many of the best commercial varieties are being propagated under controlled conditions by nurserymen from virus-free plants furnished by the U.S. Department of Agriculture and some of the state experiment stations. The nurserymen make every effort to keep these selections free from later infection, and spot tests are made before fields are certified. If the grower wishes to increase his own virus-free plants, he should start with certified plants and grow a "mother bed" at some distance from his bearing plantings and should see that all wild strawberries are eliminated from neighboring fields and fence rows. New plantings from such beds probably will remain virus-free or with little effect from the virus for several years in the bearing beds. The mother bed can be protected from virus infection only by extensive insect-control methods. It would be advisable, therefore, for the grower to plant the mother bed from certified plants each year.

Most standard commercial varieties show little evidence of the virus diseases and consequently may be used when virus-free plants are unavailable. The grower should avoid those varieties known to be especially susceptible to any of the virus troubles. For example, in the Pacific Coast region of the United States and Canada, Marshall and nearly related varieties should be avoided.

Minor Strawberry Virus Diseases

Witches'-broom. This disease was described by Zeller in 1927 from the Willamette and Hood River Valleys of Oregon [*Phytopathology*, **17**: 329–335 (1927).] It was found mainly on Marshall and Ettersburg 121. On the Ettersburg a pronounced bushy upright growth of the leaf petioles gives a "brooming" effect. It is transmitted by *Capitophorus* *fragaefolii.* It is of minor importance even where present since it can be easily recognized and diseased plants rogued.

Leaf Roll. Downward rolling of the leaflets characterizes this disease. In advanced stages the opposite margins of leaflets may touch, thus forming a tube. The leaf surface is rugose, pale green in color, with irregular yellowed areas. The disease is of little economic importance. It has been reported from Maryland, New Jersey, New York, and Vermont in the United States and from Canada.

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Chlorotic Phyllody. Plakidas in 1951 described a disease of strawberries in Louisiana which was characterized by chlorosis of the foliage and phyllody of the blossoms. The petals and carpels on the flowers were transformed into small leaflike structures with spindly petioles about ¼ to 1 in. in length. A loose rosette thus replaced the normal blossoms. In other cases the petals enlarged to form stipulate, puffed, bladderlike structures. He was of the opinion that the symptoms suggested aster yellows. In 1953 Frazier and Thomas reported on a similar disease in California and proved that the western aster yellows virus would produce symptoms similar to those observed and that the virus from phylloid strawberries could be transmitted to plantain and asters by the use of leaf hopper vectors. Thus it appears certain that the aster yellows virus is responsible for the condition known as phyllody in strawberries. The disease is of minor importance.

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June Yellows

A viruslike condition variously known as June yellows, leaf variegation, yellow leaf, gold leaf, noninfectious chlorosis, and Blakemore yellows is of considerable interest because of its puzzling character and confusion with the true virus yellows complex. It is now generally accepted that June yellows is of genetic origin.

Symptoms. In different varieties and climatic conditions symptoms vary greatly. The most pronounced symptoms occur in the early part of the year and in some instances may disappear entirely, later in the summer. At a distance, the yellow plants in a row are in sharp contrast to the deep green of normal leaves. Sometimes these plants are grouped in a well-defined section of the row, while in other cases only a few scattered plants may show the yellow foliage. The degree of yellowing varies from complete chlorosis of the leaflets to indistinct interveinal mottling of a greenish-yellow color. Variation is great, depending upon variety, season, and region. Under some conditions a definite streaking of alternate light and dark bands is evident, especially soon after the leaflets unfold. Badly affected plants become stunted and unproductive, depending on how far the yellowing has progressed. Affected plants do not recover, but may live for several years, becoming more stunted each year. Yellows plants produce only variegated runner plants.

Cause. June yellows appears to be of genetic origin. If it is of genetic origin, it must be due to somatic mutations, since it has been observed that the plants of certain varieties, such as the Blakemore, may remain a normal green through several generations but eventually give rise to yellows plants. This "continuous" mutation seems to be much more pronounced in some varieties than in others. In fact, June yellows is not known to occur in many of the standard commercial varieties. Seedlings obtained from crosses of normal green parent plants may show a certain percentage of yellows plants.

Some varieties such as Howard 17 have been found to mutate much more readily than other standard varieties. Blakemore is a variety which has caused considerable concern on the part of growers. After it became a leading variety, growers throughout the regions where this variety was grown complained of its tendency to produce June yellows plants. Attempts were made to find plantings free from yellows, and certain selections were obtained which, over a period of 6 or 8 years, showed no indication of mutation. In all cases, however, yellows finally appeared in these selections.

Hosts. June yellows has been observed in a number of varieties of the cultivated strawberry and in several wild species such as *Fragaria vesca.*

Control. Breeding new varieties has been complicated by the fact that such varieties may remain free of June yellows for a number of generations, but later may produce a certain percentage of yellows runner plants. It has been found, however, that the parent plants of certain varieties are less likely to give rise to yellows progeny than others. Progress is being made in the selection of varieties in which no variegation has appeared over a number of years. This appears to be the only method of control. Of course, varieties in which yellows has never been found may be selected if they are suitable from a market standpoint.

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Strawberry Diseases Incited by Nematodes

A general description of nematodes and the types of diseases they produce is given in Chap. 1. Strawberries are affected by several nematodes common to other fruit crops, but in addition, two important diseases in the United States and one in England are of particular interest.

Spring Dwarf. This disease has been referred to as "crimp," but this term has also been applied to summer dwarf, which is caused by a different species of nematode and has a different climatic relationship.

History and Geographic Distribution. Spring dwarf was first recognized in Massachusetts in 1932. It was considered to be of recent introduction and was regarded as probably identical with a similar disease in England called "red plant."

Spring dwarf has been reported from the eastern United States, from Massachusetts southward to Florida and Texas. It is reported also from Michigan, California, and Washington where it was probably introduced on plants obtained from the East. It is probable that the nematode has been distributed throughout the strawberry-growing regions of North America in infested plants.

Economic Importance. Severely affected plants set no fruit and are worthless. The loss of plants in any region is not large unless the growers have obtained plants from beds having a high per cent of diseased plants. Since the disease has been recognized by plantgrowers and nursery inspectors, there has been a decided reduction in the total loss from the disease.

Symptoms. Diseased plants may be recognized in early spring by the abnormal character of the crown. The bud is small and poorly developed. The hairs covering the unfolding buds are greatly reduced in number, and the stipules are more reddish than normal. The development of foliage is retarded. As the leaves unfold and elongate, they are seen to be distorted and abnormal. The petioles are swollen and the leaflets narrow and thickened. When fully formed, the leaves are small, the petioles short, and the narrow leaflets are crinkled and often curled. Sometimes the leaves fail to develop at all and the plant dies, or a few distorted leaves are produced from the lower part of the crown. Lightly affected plants may have normal-sized leaves but showing some crinkling. Such plants develop runners earlier than normal. The symptoms become less noticeable as the season advances, and lightly affected plants appear normal by midsummer. Runner plants from diseased mother plants usually become diseased since the nematodes are carried out in the buds that terminate the runner.

Causal Organism. Spring dwarf is caused by one of the parasitic nematodes, *Aphelenchoides fragariae* (Ritzema-Bos) Christie. It is a worm-shaped organism about 1 mm in length and invisible to the naked eye.

Hosts. This species has been reported on many unrelated hosts.

Disease Cycle. The nematode causing the disease is an ectoparasite; that is, it lives outside the plant tissues. Apparently this eelworm spends its entire life in the crown of the plant unless washed off or otherwise mechanically removed. In its adult form it is found in the bud between the embryonic parts and later between the bases of the petioles where it persists throughout the year. The disease is initiated by the feeding of the nematodes on the young tissue. If only a few nematodes are present, there is little effect, but when these are numerous, much of the tissue is penetrated by the stylets of the eelworm, thus causing the abnormal growth. The nematodes are carried out on the buds of the runners and thus become established in the new plants.

Distribution from plant to plant may occur when the nematodes are washed off the plant by heavy rains and carried in the water to neighboring plants. A few diseased plants in a field may result in extensive infestations.

Spring dwarf, in contrast to summer dwarf, is a cool-weather disease. The most severe symptoms appear early in the spring, and the plants are affected much less during the summer. See page 9 for life cycle of nematodes of this type.

Control. See page 449 for discussion of Nematode control.

Summer Dwarf. Summer dwarf, as the name indicates, is a hot-weather disease. In many respects it resembles spring dwarf and, in fact, was regarded for some time as a southern form of "crimp."

Summer dwarf is more common in southern regions than is spring dwarf. It is found chiefly in the southeastern and Gulf states, but has been reported from California and Washington in the West and from Illinois. It has an earlier history than has spring dwarf, since it was known to be common in Florida in the early part of the present century.

Symptoms of summer dwarf are almost identical with those of spring dwarf. The main difference is in the time of appearance. Summer dwarf shows its most conspicuous symptoms from about July 1 to October 1. In the spring the foliage does not appear to have any of the characteristic symptoms of dwarf. On this account, the outer, older leaves are more or less normal, with the inner and younger leaves crimped and distorted.

The *causal organism* is *Aphelenchoides besseyi* Christie.

The life cycle and method of dissemination of the nematode are much the same as that of *A. fragariae.*

HOSTS. Nematodes identified as *A. besseyi* have been reported as the cause of white tip of rice and of a yellow bud blight of vanda orchids. Nematodes from the orchids were transferred experimentally to African violets. It is not clear, however, whether nematodes from those plants are able to cause disease in strawberry.

Cauliflower Disease. In England a disease of strawberry similar in many respects to summer and spring dwarf in North America has long been known, having been described by Ritzema-Bos in 1889. He considered it to be caused by a nematode which in 1891 he named *Aphelenchus fragariae.*

In 1952 Crosse and Pitcher published experimental results which indicated that the cauliflower disease as found in England was the result of a relationship between a nematode, *Aphelenchoides ritzema-bosi,* and a bacterium, *Corynebacterium fascians.* Earlier workers had concluded that the nematode alone was not responsible, since attempts to produce the disease by inoculation gave erratic results, and, furthermore, the nematode could often be found in abundance in normal crowns of the strawberry.

Crosse and Pitcher stated that considerable variation in symptoms occurred from a "red plant" open-crown condition to the fully developed "cauliflower" type. Also, variations in enation, secondary crowns, and alaminate leaves were observed. They found by inoculation that the typical cauliflower condition resulted when either of two strains of the bacterium, *C. fascians* and the nematode *A. ritzema-bosi,* were introduced into the crown. In some cases the eelworm alone gave fairly typical cauliflower, but it was found that the bacterium could be isolated from these crowns, and they concluded that in nature this bacterium was often present. The bacterium alone would not produce the symptoms, and only certain strains were effective when used in combination. They found that another species of nematode, *A. fragariae,* was commonly associated with the "red plant" disease. This species was also found in cauliflower plants, but whether the bacterium or the other eelworm species was present was not determined. Their final conclusion was that "cauliflower is simply the least frequent of a range of symptoms of a bacterial disease endemic in strawberry plants infested with *Aphelenchoides* spp."

The relation between cauliflower, red plant, summer dwarf, spring dwarf, and the interrelation of these diseases with associated bacteria are still obscure.

Christie in 1942 in describing the summer dwarf nematode *A. besseyi* stated that nematodes obtained from typical cauliflower plants in England were definitely different from this species and more closely resembled the spring dwarf nematode, *A. fragariae.*

A. ritzema-bosi, as Allen wrote in 1952, also occurs in the United States.

Root Knot. Root knot of strawberry is caused chiefly, if not entirely, by *Meloidogyne hapla* Chitwood, the northern root knot nematode. When Sasser tested four species and a variety of *Meloidogyne* on strawberry, this was the only one that developed at all, and it thrived in all four varieties of strawberry in his tests. Fortunately, *M. hapla* is the species that is most readily controlled by crop rotations because it does not develop in roots of corn or the small grains.

Plants attacked by root knot show the usual knotlike galls on the roots, accompanied by reduced growth of main roots and excessive production of short branch roots, which, in turn, may be infected. Stunting and decreased production occur from light to moderate infections; very severe attacks may contribute to death of the plants under adverse conditions. As is generally true with other crops, root knot is less commonly troublesome in heavy soils than in sandy or muck-type soils.

For further discussion of the root knot nematodes, see Chap. 1 and also under Root Knot of stone fruits. Information on control is given on page 449.

Other Nematodes on Strawberry. Among other nematodes known to attack strawberry are the root lesion or meadow nematodes, *Pratylenchus* spp. At least three species have been reported from this crop, *P. vulnus*, *P. brachyurus*, and *P. pratensis*. Such nematodes cause necrotic lesions in the root cortex, contributing to the cortical root rot complex. Although they occur widely in agricultural soils and are being distributed in nursery stock, it is not clear how much damage they do to the strawberry. For further information on the root lesion nematodes, see Chap. 1 and also under Stone Fruit Diseases.

Control of Nematodes Attacking Strawberries. VARIETAL RESISTANCE. Christie, in 1928, reported on the susceptibility of 36 varieties of strawberries to the southern dwarf nematode, *Aphelenchoides besseyi* (called a strain of *A. fragariae* in his report). He found all 36 varieties susceptible in varying degrees. No evidence seems to be available concerning possible resistance to other nematodes. It seems, therefore, that at present varietal resistance cannot be depended upon for nematode control.

HOT-WATER TREATMENT. It has been found possible to rid strawberry plants of endoparasitic nematodes in roots by immersion in hot water without serious injury to the plants. The same treatment should greatly reduce, if not fully eliminate, the ectoparasites of the bud. This method is used mainly to rid the mother plants of nematodes for establishing them in nematode-free soil for multiplication, but may be used on a much larger scale by nurserymen to assure growers that there is no danger of introducing nematodes in their plantings. State-wide programs in cooperation with the U.S. Department of Agriculture have been set up for furnishing strawberry growers nematode-free plants. However, it is essential that these plants be planted in fields where the nematode population is low.

Rather rigid requirements are necessary for satisfactory results from the hot-water treatment. Accurate control of temperature and time is essential, as is also the condition of the plants and their aftertreatment. The plants should be in a dormant condition, that is, winter-dug and cold-stored. From cold storage they should be immersed in the hot water maintained at 127°F for 2 minutes, then surface-dried and returned to cold storage until time to plant, allowing a minimum period of several days before removal from storage. For the treatment of foliar nematodes, the plants should be immersed for 7 minutes at 171°F.

SOIL TREATMENT. A general discussion of soil treatment for nematode control is given in Chap. 1. The nematode parasites of strawberry roots are able to develop freely in various other crop plants and weeds, with the result that a new field may already be generally infested before strawberries are planted. There is little point in trying to obtain nematode-free nursery stock if the plants are to be set into infested soil.

Chloropicrin and mixtures containing methyl bromide, chlorobromopropene, ethylene dibromide, or dichloropropene are recommended for soil fumigation. All are good insecticides and nematocides, and some give a measure of control of fungi. Special types of applicators have been devised for soil treatment where large-scale fumigation is necessary.

Choice of fumigant, dosage, time of application, soil condition, and other factors must be considered in soil fumigation. Since these vary in different localities, the grower should consult his local agricultural experiment station for detailed recommendations. Allen and Raski reported satisfactory control of root lesion nematodes in strawberry by means of split applications of either of two commercial formulations: CBP-55 (technical chlorobromopropene), at the rate of 30 gal per acre, and D-D at 40 and 80 gal per acre. Soil fumigation is costly and should not be undertaken unless the soil to be used is known to be heavily infested or unless an attempt is made to grow nematode-free plants.

Crop Rotation. Although crop-rotation programs sometimes are not feasible on land of very great value, the possibilities for nematode control by suitable rotations are better with strawberry than with other fruit crops. This results in part from the shorter period of years that one planting is to be kept in production, by comparison with the orchard crops, and in part from the host limitations of the nematodes themselves. The bud nematodes that cause the dwarf diseases apparently do not thrive in any crop likely to be rotated with strawberry and do not survive long in a fallow field. The important root knot of strawberry is caused by *Meloidogyne hapla*, a species that does not develop in cereal crops or, apparently, in cucurbits with the exception of muskmelon. In southern areas, the immune *Crotalaria spectabilis* also is available as a cover crop. Growth of any of these resistant plants for a time before planting strawberries, or between plantings, will help to reduce the root knot nematode infestation, provided that susceptible weeds are not allowed to develop. The same cropping may not, however, be of any help in control of root lesion nematodes, of which there are several species with host ranges still poorly defined.

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CHAPTER 11

DISEASES OF GOOSEBERRIES AND CURRANTS

Gooseberries and currants are members of the genus *Ribes* of the family *Saxifragaceae.* Native species of both are found in the northern latitudes of the North Temperate Zone in Asia, Europe, and North America and at high altitudes southward. Cultivated currants and gooseberries are derived either directly from these wild species or by interspecific crossing. Both were mentioned as common garden fruits in northern Europe as early as the sixteenth century. European currants and probably gooseberries were brought to the New World with the earliest colonists (around 1629). White and red currants in America are mainly derived from the European species *Ribes sativum* and *R. rubrum* or hybrids of these two species. Black currants (*R. nigrum*), while extensively grown in northern Europe, are not grown to any extent in North America. A Norwegian currant species *R. petraeum* is of special interest since it was from this species that the Viking variety, resistant to the white pine blister rust, was derived.

The European gooseberry *R. grossularia* is the source of most cultivated gooseberries in Europe, and it thrives in North America, but because it is very susceptible to powdery mildew, an American species *R. hirtellum* was utilized for breeding commercial varieties in the United States.

Several species of *Ribes* are also used for ornamental planting, and the wild, usually spiny gooseberry *R. cynosbati* and the smooth-fruited species *R. hirtellum* were used by early settlers in North America for jelly and jam.

Both currants and gooseberries are much more extensively utilized in Europe than in North America.

Currants and gooseberries are subject in common to a number of important diseases, but usually vary as to susceptibility to individual diseases.

Anthracnose of Currants and Gooseberries

History and Distribution. Anthracnose, also called "leaf spot" by English authors, was first described from Europe in 1867, but since it was reported in America six years later, there is some doubt as to its original home. Since these early descriptions, the disease has been reported from most of the countries where species of *Ribes* are grown.

Anthracnose was reported on *Ribes rubrum* (red currant) from Flanders by Kickx in 1867. Berkeley identified the disease on *R. nigrum* (cultivated black currant) from America in 1873. It was noted as a serious disease in New York by Dudley as early as 1889, but did not receive much attention either in America or Europe until near the close of the century, when it appears to have caused serious injury in several countries. Klebahn discovered the perfect stage and named the fungus *Pseudopeziza ribis* in 1906. Between 1905 and 1913, Ewart reported on a series of experiments on the fungus, especially in relation to infection and spore germination, and on control. A comprehensive study of the physiology and life history of the fungus was reported by Blodgett in 1936. He called special attention to the existence of variation in a number of isolates, especially as to their pathogenicity.

Economic Importance. Pathologists consider anthracnose the most serious gooseberry and currant disease. Before these crops were grown extensively on a commercial scale, the importance of the disease was not realized. Injury to the entire plant as a result of defoliation is evidenced by the reduced size of the fruit and the limited growth. Fruit infections also occur, thus further reducing the quality of the crop. Various authors have reported losses of from 50 to 80 per cent as a result of severe infections. Over a period of years the losses from this disease may not be great, owing to the fact that it is only under conditions unusually favorable for the fungus that complete defoliation or fruit infection occurs. In Illinois it was observed that over a period of 8 years, defoliation was severe in 4, mild in 2, and very light in 2 years. In some regions the disease is most destructive to currants, while in others the gooseberries suffer more damage. For example, the disease is reported as worse on currants in Wisconsin, while in Illinois and Indiana the main loss is to the gooseberries. This may be due in part to differences in the strains of the fungus present in these areas.

Symptoms. The symptoms vary with the host plant attacked. All succulent parts of the current season's growth are susceptible, but the *leaf spotting* is the most pronounced symptom. Usually, numerous dark brown to black dots appear scattered at random over either or both surfaces of the leaf (Fig. 90). They may appear at any time during the growing season, from shortly after the opening of the leaves to late fall. These spots enlarge somewhat, become more angular in outline, and on some hosts are bordered by a purplish area. Shortly after infection, especially in severe cases, the entire leaf blade turns yellow and defoliation occurs. Often there is a green zone surrounding each spot in these yellowed leaves. The spots vary in size, especially on different hosts. On the gooseberry, they are about ⅛ in. in diameter, but often are so numerous as to coalesce, in which case the dead area involved may be much larger. The extensive yellowing of the leaves is the most characteristic symptom of the disease on the gooseberry, but is not so pronounced on the currant. Shortly after the spots become evident, the spore masses may be seen as glistening, dull gray, hemispherical, slimy droplets. These are most evident during a period of high humidity or following a rain. They may be observed on either surface and vary in number from one to ten or more to the spot. Under some conditions the spore masses project from the leaf in the form of short, whitish tendrils.

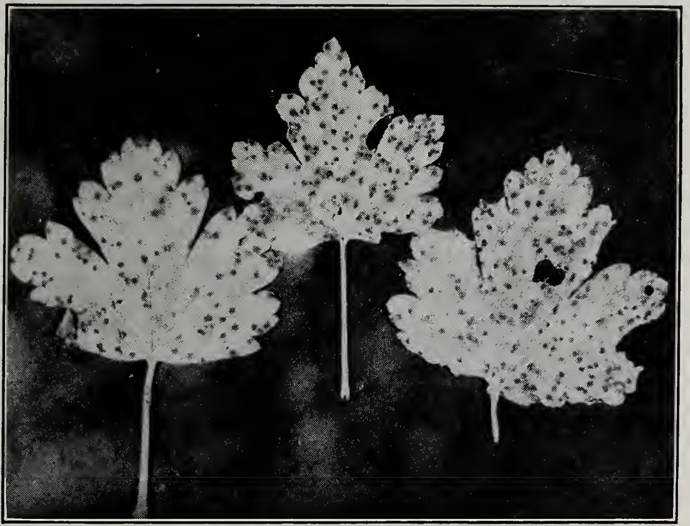

Fig. 90. Anthracnose on gooseberry leaves. Yellowing apparent on left.

On the *petioles* of the leaf and on the *pedicels* of the fruit the lesions are somewhat elongated, often girdling the organ attacked. On the *fruit* the spots resemble flyspecks. On the *stem* of young shoots the superficial lesions are usually larger than on the leaves and light brown in color, rarely producing spore masses.

Causal Organism. *Nomenclature.* The fungus causing anthracnose is an Ascomycete which produces an open cuplike ascocarp and consequently belongs in the order Pezizales. Montague and Desmaziers described the imperfect stage of the fungus as *Gloeosporium ribis* in 1867. In 1906, Klebahn discovered the perfect stage on *Ribes rubrum* and *R. nigrum* and named it *Pseudopeziza ribis.*[1] He was able to prove the connection both by inoculation with the ascospores and by comparison of cultures obtained from the two types of spores. His findings were substantiated by Blodgett, who found the perfect stage in America both on currants (*R. rubrum* and *R. americanum*) and on the gooseberry (*R. grossularia*). In 1933 and 1934, he secured typical infections with ascospores and comparable growth in culture to that produced by the conidia.

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[1] Class Ascomycetes, Order Pezizales, Family Mollisiaceae.

Hosts. So far as known, this disease occurs only on members of the genus *Ribes*. Both cultivated and wild species of gooseberries and currants are susceptible, and no species or variety of either is known to be immune.

Morphology and Cultural Characters. *P. ribis* produces three types of spores: ascospores, conidia, and microconidia. The *apothecia*, in which the asci are borne, are found on the dead leaves in early spring. They are cup-shaped, later expanding to saucer-shaped, dark brown, and fleshy, and borne on a very short stalk partly embedded in the leaf tissues. They measure about 0.5 to 1 mm in diameter but vary greatly in size. The *asci* are clavate and borne among the simple, rarely septate paraphyses. The *ascospores* from currant (*R. rubrum*) averaged 15.5 by 6.7 μ, while from gooseberry they averaged 20.2 by 9.3 μ, according to Blodgett. They are hyaline, nonseptate, and ovoid in shape.

The *conidia* are borne in *acervuli* which are scattered over the lesions on both leaf surfaces. The spores form hemispherical, grayish, glistening masses, which are viscid but easily dissolve in water. The conidia are, in general, crescent-shaped, but vary considerably as to the amount and type of curvature. Measurements from point to point across the crescent averaged 17 to 24 by 5.3 to 5.8 μ, while the actual length is from 28 to 36 μ, according to Blodgett. He found that spores produced in culture were consistently larger than those from leaves. The conidia are hyaline, granular, nonseptate, but may become uniseptate during germination. *Microconidia* are formed both in culture and on the host. The microconidia are hyaline, nonseptate, bacilloid in form, and vary greatly in size. The length may vary from 5 to 11 μ and the width from 1.4 to 2.3 μ. The function of the microconidia is not known, but it is suspected that they may have some relation to the development of the perfect stage, as is the case with microconidia of other Ascomycetes.

In *culture*, the fungus grows slowly, producing on dextrose agar a growth of about 1 inch in 8 weeks. Both conidia and microconidia, but not ascospores, are produced. Optimum temperature for growth is between 60 and 70°F.

Disease Cycle. Primary infection in the spring may be initiated by ascospores or conidia. Conidia may be formed in abundance on the old leaves in the early spring, and it has been demonstrated that conidia may survive the winter and retain their viability. Ascospores are forcibly discharged from the old leaves beneath the bushes and may be carried by currents of air to the unfolding leaves. Conidia are formed in viscid masses and consequently must depend on water to dissolve and disseminate them. Usually, infection is first noticed on the lower leaves on which the conidia could be easily splashed during rains. Since conidia may be formed on overwintering leaves when the temperature is above freezing, infections may occur at any time, when susceptible parts, such as unfolding leaves, are exposed and sufficient moisture is present. Ascospores are formed and are mature early in the spring, so that these also may bring about early primary infection. Secondary infection takes place throughout the season from the conidia produced on the green leaves. Often, following complete or partial defoliation, new terminal growth occurs, and this in turn may become heavily infected.

After the leaves fall, the fungus starts a saprophytic growth in the leaf tissues resulting in a widespread development of fungus mycelium beyond the confines of the original lesions. Heavy mats of pseudoparenchymatous tissues are formed, from which the initials of fruiting bodies may arise.

Relation to Environment. Anthracnose seems to become prevalent under a wide range of conditions, but it requires fairly moist spring conditions for abundant infection.

Germination of conidia and ascospores may occur at any temperature between 35 and 82°F, with an optimum for conidia between 55 and 70° and for ascospores between 40 and 60°F. Germination does not occur above 85°, so that high summer temperatures are unfavorable for infection. A moist period of from 12 to 24 hours is sufficient for infection.

Host Relations. Infection may occur on both the under and upper surface of the leaf. Penetration is directly through the cuticle and epidermal cell wall. Within the epidermal cell, the penetration tube expands into a bulbose structure from which arise several fine hyphal branches which may completely fill the epidermal cell. The secondary growth produces slender hyphae which extend into the intercellular spaces and cells of the palisade layer, finally invading all the tissues of the leaf. A heavy growth of the mycelium just beneath the epidermal cells occurs, and from this mass the acervuli arise. Penetration by germinating ascospores and subsequent development are much the same as for the conidia.

The cells of the tissue attacked soon die and become disorganized. Since the mycelium is localized in the region of the lesion, it is evident that some toxic material must be produced to account for the subsequent chlorosis of the entire leaf.

Control. *Sanitation.* Since the fungus lives over winter beneath the bushes, the removal of old leaves before spring would eliminate much of the inoculum. This procedure is not practical in large plantings, especially in the case of gooseberries. In young plantings of both currants and gooseberries, this practice would be more practical.

Varietal Relationship. It is generally agreed that all varieties of both currant and gooseberry are susceptible to anthracnose. Conflicting reports of relative susceptibility of varieties of *Ribes* are given by various investigators. Blodgett states: "From such reports in literature and from studies and observations by the writer, it is concluded that all commonly cultivated varieties of red, white, and black currants and gooseberries may be infected by *Pseudopeziza ribis*, their comparative susceptibility being influenced largely by environmental conditions."

Under Illinois conditions, gooseberries in general have been found to be much more susceptible than currants. Over a period of 4 years, the percentage of defoliation of some 25 varieties of gooseberries was recorded. Of 16 selected varieties, 9 showed severe defoliation (60 to 100 per cent) three out of four seasons (Triumph, Downing, Haughton, Josselyn, Columbus, Oregon Champion, White Smith, Portage, and Pearl). Carrie, Como, and Transparent showed only light or medium infection. Poorman, very generally raised, was most erratic in its behavior, showing severe defoliation in 2 years, light in one, and medium in another.

Colby crossed a number of standard varieties of gooseberries and, from a study of the seedlings, came to the conclusion that those in which Transparent was used as a parent showed the highest degree of resistance, but all were susceptible.

Spraying. As early as 1889, Dudley suggested the use of copper sprays for the control of anthracnose. Ewart in Germany secured excellent control with various copper-containing sprays. Bordeaux mixture has been used successfully in a number of places for control and has not caused serious injury. Lime sulfur has also given fair control of the disease and has the advantage of controlling mildew on gooseberries when this is present. At high summer temperatures, the sulfur sprays may cause injury, and for this reason it is best to apply them early in the season and Bordeaux mixture after the temperature is likely to be high during the day. The first spray should be applied shortly after the leaves appear. The number of applications necessary for practical control will depend upon weather conditions. From a practical standpoint, it is not necessary to continue the sprays late in the season, in spite of the fact that the disease may become so serious as to cause partial defoliation. A number of the organic fungicides have given good control, but some of these cause trouble when the fruit is canned.

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Cane Blight of Currant

History and Geographic Distribution. Cane blight was first reported from the Hudson Valley in New York in 1891. The causal organism was not determined at this time. In 1900, Duggar started a study of the disease and the causal agent. Grossenbacher and Duggar later continued the study and issued a comprehensive publication in 1911 on the life history and parasitism of the causal organism which they named *Botryosphaeria ribis.* Further contributions were made by Stevens and Jenkins in 1924 on the relation of the pathogen to the same organism occurring on other hosts, especially the rose. They confirmed Duggar's statement that there were two forms of the fungus, one chromogenic, which was an active parasite, while the nonchromogenic form did not produce the typical blight.

The interest in the disease on currants waned, but the fact that *B. ribis* caused a serious disease of roses and apples led to further studies of the fungus.

Cane blight has been reported on currants and gooseberries from most of the states where these crops are grown but seems to be most prevalent in the eastern and northern states. Absence of reports from outside of the United States indicates that it is probably of American origin, possibly indigenous on some hosts other than the currant.

Economic Importance. At one time cane blight was considered a very destructive disease of the currants. In New York some long-established plantings showed from 25 to 50 per cent of the bushes dead from blight. Once established the disease soon spreads to all the canes of a bush, and thus there is a total loss of crop on infected bushes. The disease is now of minor importance, since most commercial growers have learned how to control it by cultural methods.

Symptoms. Evidence of cane blight is the sudden wilting and dying of scattered *canes* or whole *bushes* during the summer months and is most conspicuous just before the fruit ripens. The wood and pith of these diseased canes have a blackened appearance. Later in the season on the current season's growth or on canes not removed the previous season, black wartlike bodies arranged in rather definite parallel rows appear on the diseased canes (Fig. 91). Often another fungus having brick-red fruiting bodies appears on these canes. This is a *Nectria*, which is not regarded as parasitic.

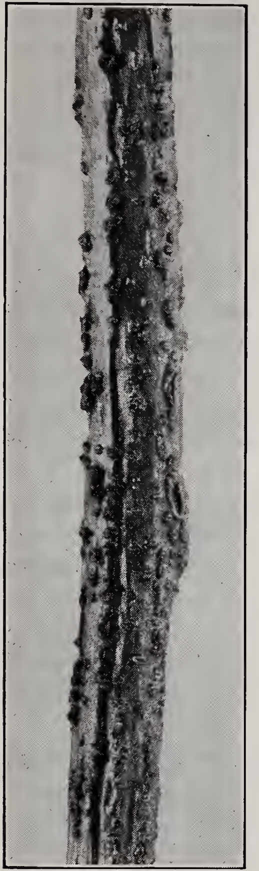

FIG. 91. Fruiting bodies of currant cane blight on 2-year-old canes.

Causal Organism. An Ascomycete *Botryosphaeria ribis* Gros. and Dug.,[1] is the fungus causing cane blight. Two types of imperfect spores are produced. During July simple *pycnidia* are formed on the withered tips. This is called the *Macrophoma* stage. Later in the season stromatic bodies are formed along the stem in which the *Dothiorella*-type spores are produced. In the late fall and winter the perithecia of *Botryosphaeria* are formed in the stromatic bodies, which enlarge and become wartlike in appearance. The pycnidia of the *Macrophoma* are globular, 175 to 250 μ in diameter. The *spores* are fusoid, continuous, hyaline, and measure 16 to 25 by 4.5 to 7.5 μ. The pycnidia of the *Dothiorella* stage are borne in the stroma, often interspersed with perithecia in the same stromatic structure. The spores are similar to the *Macrophoma* spores. The perithecia are borne in the same or similar stromata, usually appearing in late summer. When the perithecia mature, the ostiole is much more prominent than that of the pycnidia, and the stromata bearing the perithecia are studded over with these projecting structures. The ascospores are continuous, fusoid, hyaline, 16 to 23 by 5 to 7 μ.

As mentioned earlier, Grossenbacher and Duggar found two strains of this fungus and determined that one strain always produced a pink to red color when grown in starchy media. The achromogenic form which is not parasitic is indistinguishable morphologically from the pathogenic chromogenic strain. This was confirmed later by other workers.

Hosts. *B. ribis* is most commonly found on the red currant but has been reported as occurring on a number of species of *Ribes*, both currant and gooseberry. It causes an important disease on rose canes and was isolated by Stevens from horse chestnut. It occurs commonly on dead apple twigs and causes a fruit rot and canker on this host (see page 184).

Disease Cycle. The fungus passes the winter in the blighted canes. In the spring ascospores are discharged and infect the developing shoots. Pycnidia ooze spores through the early summer months which are carried by various agents to the new growth. After infection occurs the mycelium grows downward through the bark, wood, and pith. If infection starts on a lateral shoot, as if often does, the fungus grows downward into the main stem where it encircles and kills the cane. A cane-boring insect is reputed to be of some importance in the dissemination of the spores and in initiating infection.

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[1] Class Ascomycetes, Order Dothidiales, Family Dothidiaceae.

Control. Cutting out and burning of diseased canes has proved fairly effective in keeping down the disease to a point where no great loss occurs. Care should be taken in planting to secure cuttings from bushes known to be free of the disease.

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Stevens, N. E., and A. E. Jenkins: Occurrence of the currant cane-blight fungus on other hosts. *J. Agr. Research,* **27**:837–843 (1924).

American Powdery Mildew of Gooseberry and Currant

This disease is referred to as "American" powdery mildew in order to distinguish it from the much less destructive European powdery mildew caused by a related species. While most important as a disease of the gooseberry, it occurs in a milder form on red, white, and black currants.

History and Geographic Distribution. As the name implies, this mildew is of American origin, being native on wild species of gooseberry. It is believed to have been carried to southwest Russia in 1890. It was reported from Ireland in 1900, probably having been independently introduced from America. In Europe it soon became generally established at the beginning of the century and was apparently introduced into England from the Continent where an outbreak in 1906 was recorded.

American mildew is known to occur in most countries in the North Temperate Zone where species of *Ribes* are grown. It has not been reported from the South Temperate Zone.

Economic Importance. Most damaging to gooseberries in North America, mildew is of minor importance except on certain susceptible varieties, especially those recently imported from Europe. In the British Isles and in the European Continent severe losses may occur. The thick, webby growth of the fungus over the surface of the leaves and shoots, while superficial, greatly retards photosynthesis and thus stunts the plant. The developing fruit is also often covered, resulting in dwarfing, roughening, and often cracking, so that it is worthless.

Symptoms. The lower parts of the bush usually show the first signs of infection. In May or June small superficial white patches first appear on the *leaves*, *shoots*, and *berries*. These spots soon enlarge and coalesce to form large patches. The leaves and tips of the shoots may become distorted. The infected patches take on a white, dusty appearance due to the production of great numbers of conidia. These patches later turn to a rusty-brown color as the superficial layer of mycelium ages. The affected fruit is discolored and has a roughened surface. The thick weft of brown mycelium remains on the shoots after the leaves are shed. The black perithecia may be seen as very small dots in the mycelial patches.

Causal Organism. The American mildew fungus is one of the Erysiphaceae (powdery mildews). *Sphaerotheca mors-uvae* (Schw.) B & C.[1] The imperfect, or *Oidium*, stage is the one responsible for the white powdery appearance of the patches. The *mycelium* is entirely superficial, but like other powdery mildews offshoots from the hyphae penetrate the epidermal cells, forming haustoria, from which the fungus derives its nourishment. An unusually thick mat of brown mycelium is formed by this fungus later in the season.

Arising from the mycelial mat are numerous, club-shaped conidiophores on which are born chains of powdery conidia. These *conidia* are oval to oblong, hyaline, and easily detached after maturity.

The *perithecia* are closed (cleistocarpic) as in other powdery mildews. They are dark-walled, subglobose, 76 to 100 μ in diameter. The *appendages* arising from the perithecium are few in number (sometimes absent), filamentous, flexuose, and with no distinct terminal branching. Only a single large *ascus* is produced in each perithecium. This contains eight ellipsoid hyaline *ascospores* measuring 12 to 15 by 20 to 25 μ.

Disease Cycle. Both conidia and ascospores may cause infection. In the spring the perithecia are ruptured by the swelling asci, and the ascospores are forcibly discharged. The perithecia are abundant on the pruned or broken twigs on the ground, and for this reason infection often appears first on the lower leaves and shoots. Conidia are soon formed on the initially infected spots and are carried by air currents and washing to expanding organs, including the fruit.

Unlike grape powdery mildew which seems to be favored by dry periods, gooseberry mildew is especially destructive in areas having cool, humid, and rainy periods during spring and early summer.

Control. Considerable difficulty has been experienced in England and Ireland in checking this disease. Lime sulfur is effective, but some varieties are sulfur-sensitive. Dusting sulfur is fairly effective if applied often.

Spacing bushes to allow quick drying and cleaning out all debris from under the bushes will reduce chances of infection.

A number of resistant varieties have been listed from time to time, but some of these seem to be quite susceptible in some regions, probably because of biologic strains of the fungus. In Austria the Höflein variety is reported as being very resistant. In England Butler reports Lancer, Crown Bob, and White Smith as fairly resistant. Some resistant American varieties when grown in Ireland were found to be susceptible.

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[1] Class Ascomycetes, Order Perisporales, Family Erysiphaceae.

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European Gooseberry Mildew

Another powdery mildew fungus belonging in the Erysiphaceae occurs on gooseberries and currants but is much less important than the American gooseberry mildew. It occurs mainly on the upper surface of the leaves and rarely on other organs. The weft of superficial mycelium is much lighter than in the American mildew and does not form feltlike brown patches as in the latter. It has been reported from a number of localities in the United States, but appears to be much more common in European countries.

The disease is caused by *Micosphaera grossulariae* (Wallr.) Lev. In addition to the different type of growth this fungus can be easily identified by the dichotomously branched, recurved tips of the appendages of the perithecia. Also four to six asci are produced in the perithecium, instead of the one in *Sphaerotheca mors-uvae.*

White Pine Blister Rust

The common name "white pine blister rust" is generally used by American authors since it is on this valuable timber tree that the disease is most important. The common name "currant rust" is used when the disease is considered in relation to *Ribes* species, but another rust also occurs on members of this genus, so this name is not appropriate.

Like apple rust the fungus causing the disease is heteroecious, but in this case the aecial stage occurs on pines and the uredial and telial stages are on currant and gooseberry plants, while on the apple the telial stage is on the cedar and the aecial stage is the one causing the damage to the apple.

The white pine blister rust is not of great economic importance on the currant and gooseberry since even when present no great damage is caused. However, the fact that these fruits serve as alternate hosts of the fungus has resulted in restrictions on the growing of these crops in regions where stands of susceptible pines are grown. It has also involved the spending of millions of dollars by the United States government for eradication and inspection programs.

History and Geographic Distribution. The fungus on species of *Ribes* in Europe was described in the middle of the past century. It is generally believed that it spread into western Europe from some native area in Siberia, where it occurs on *Pinus cembra*. Specimens of the rust on *Ribes aureum* (flowering currant) were collected in Kansas in 1892, but it was not until 1906 that attention was called to the wide distribution of the disease in the northeastern section of the United States. At that time it was known to occur throughout Europe and undoubtedly was imported into North America on white pine seedlings at various times during the first decade of the present century.

In 1888 Klebahn established the relation between the *Peridermium* stage on pine and the *Cronartium* stage on *Ribes*.

Between 1910 and 1930 extensive studies were made on the disease in the United States as a result of its importance on the susceptible pines. Control measures were worked out to protect the white pine stands and to prevent the further spread to areas free of the disease. These consisted in the eradication of *Ribes* species within a specified distance of the pines and the prohibition of importations of pines from foreign countries where the disease was known to occur. Extensive surveys were made during this period to determine the distribution of the rust and the species of pine susceptible to the rust. Also numerous inoculation tests were made to determine the susceptibility of species and varieties of *Ribes*. In 1935 Hahn recorded the immunity of a variety (Viking) of the red currant, which had been regarded as highly resistant by Norwegian workers.

This rust is now known to occur in all countries of the North Temperate Zone where susceptible varieties of pine are present. Since the fungus cannot survive from year to year on *Ribes*, the disease is not present in regions free of susceptible pines. This is true also of areas where the distance between the pines and currants and gooseberries is more than a quarter of a mile.

Symptoms. The *uredial* stage on *Ribes* appears in the spring as small yellow spots on the underside of the *leaf*. Often the pustules are widely spaced, but may be so numerous as to occupy most of the surface. The leaf shows no particular damage as a result of the attack, although later in the season some defoliation may result. These powdery pustules continue to appear on the new growth since the urediospores can cause repeated infections. Toward the end of the summer another type of the rust fruiting structure is found. Elongated, cylindrical "horns" arise either from the old uredial pustules or independent of these. On these yellow to brown threadlike growths, the teliospores are produced in summer and early fall.

Infection on the pine results in spindle-shaped enlargements from which the aecial pustules break through, producing large quantities of yellowish aeciospores. Eruption takes place in April, May, and June, and it is during this period that the aeciospores are carried to the *Ribes*.

Causal Organism. *Cronartium ribicola* Fisch.[1] is the cause of the disease. The uredia are formed in small yellowish pustules on the underside of the leaves. The urediospores are ellipsoid, 12 to 14 by 19 to 35 μ. The walls are sparsely echinulate. The telial columns, yellow to brown, extend about 2 mm from the leaf surface and usually arise from the uredial sori. The one-celled teliospores are similar to the urediospores, measuring 8 to 12 by 30 to 60 μ.

Disease Cycle. The aeciospores from the pine alight on the leaves of the *Ribes* plants in the spring (April to June), and the germ tube penetrates the stomata on the underside of the leaf. Growth of the mycelium is mostly in the mesophyll of the leaf, and haustoria are found in the host cells along the path of the intercellular hyphae. No noticeable hypertrophy is evident, and little injury seems to result. About 2 or 3 weeks after inoculation the mycelium forms a mat of thick cells in certain areas, and from this the uredial pustules arise and push up to the surface where the urediospores are formed and liberated. These spores are then capable of spreading the disease throughout the remainder of the growing season. The teliospores are formed in the summer and will germinate to produce *sporidia* (basidiospores) as soon as mature. During moist weather the telial column may be covered by a pinkish coating of sporidia. The sporidia are carried by air currents to the pine, where they bring about infection. The fungus lives for a number of years on the pine, and consequently new crops of aeciospores may be produced each year to reinfect the currants and gooseberries.

Hosts. Most of the pines belonging to the five-needle group are susceptible. The white pine, *Pinus strobus*, called the Weymouth pine in Europe, is the one most commonly associated with this disease.

Practically all species and varieties of *Ribes* are known to be susceptible, as proved by inoculation experiments. A Norwegian species of currant, *R. petraeum*, appears to be highly resistant, and from this species the immune Viking variety originated. No hosts outside the genera *Pinus* and *Ribes* (including *Grossularia*) are known. Black and flowering currants are reported as being especially susceptible in Europe. Hahn, in 1948, reported the successful breeding of two black currant hybrids [Kerry (*Ribes nigrum*) and *Ribes ussuriense*], which were immune to blister rust. These were of good quality.

Control. No control measures are necessary for currants and gooseberries except that they should be grown at a distance from infected pines.

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[1] Class Basidiomycetes, Order Uredinales, Family Melampsoraceae.

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Cluster Cup Rust of Gooseberries and Currants

This rust, caused by *Puccinia pringsheimiana* Lagerb (*P. caricis grossulariata* Arth.), is of little economic importance. The cluster cup stage (pycnia and aecia) occurs on species of *Ribes,* while the uredial and telial stages are found on a large number of species of *Carex* (sedge). In North America it is common only on wild species of gooseberries or in neglected gardens, probably owing to the presence in these locations of the alternate sedge host.

The rust attacks *leaves, stems,* and *fruit* but is most commonly found on the leaves and petioles. The infected leaf is thickened in the area where later the cluster cups appear. These spots take on a red appearance, and later the aecial cups break through on the underside of the leaf. Some curling and distortion of the leaf results. Similar enlargements appear on the *shoots* and *petioles.* On the shoot the infected area may extend some distance along the stem. On the berries the red spots are somewhat thicker than on the leaves. These cushions become studded with wartlike projections which later break through the outer tissue to form the aecial cups. Affected fruit is worthless, but usually not many berries on a bush are infected. The cups extend well above the surface of the cushion.

The aeciospores are shed and infect any sedge that may be in the neighborhood.

Control consists in the elimination of the alternate sedge hosts in the neighborhood of the gooseberries and currants. (E. J. Butler and S. G. Jones, Cluster cup rust of gooseberries. *In* Plant pathology, Macmillan & Co., Ltd., London, pp. 821–824, 1949.)

Mycosphaerella Leaf Spot of Gooseberry and Currant

This disease is often referred to as "*Septoria* leaf spot" since the parasitic stage of the fungus is a *Septoria.*

History and Geographic Distribution. The fungus causing leaf spot was described as early as 1842. Since the disease was considered of little importance, very few early references to it can be found. It is known to be generally distributed on cultivated and wild species of *Ribes*. In the United States it is most destructive in the Mississippi Valley region, but has been reported as common from both eastern and western states, as well as from Canada and Alaska.

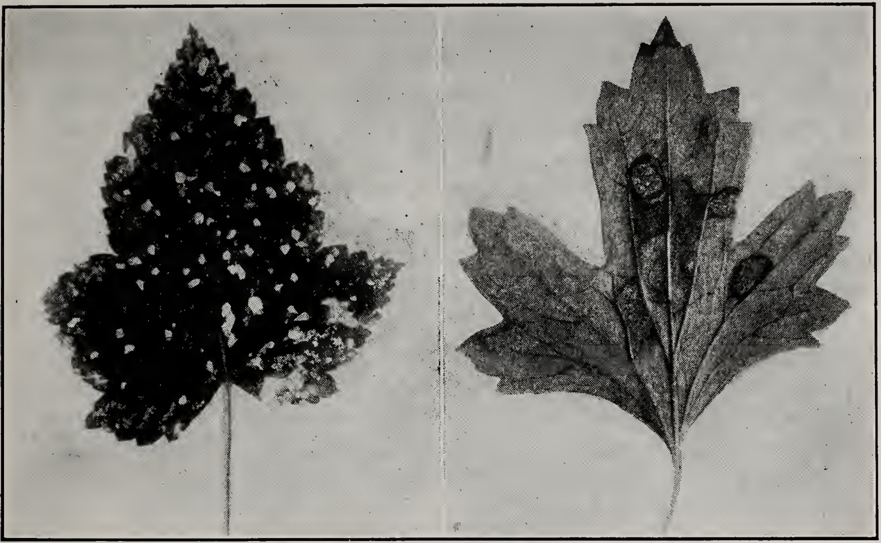

Fig. 92. *Mycosphaerella* leaf spot. At left, on black currant; at right, *Mycosphaerella aurea* on flowering currant, showing pycnidia.

Economic Importance. In general this leaf spot does not cause much loss, but extensive defoliation may reduce the vitality of the plant. When defoliation occurs early, the fruit may be sun scalded.

Symptoms. *Mycosphaerella* leaf spot is easily differentiated from that caused by the anthracnose fungus (*Pseudopeziza ribis*). About June the small brown spots appear on the infected leaf. At this time they may resemble anthracnose, but they soon enlarge and the central area becomes light in color with a brown border (Fig. 92). Small black specks are scattered over the surface of this area. The diseased leaves, especially on currants, turn yellow and drop.

Causal Organism. The imperfect stage of the leaf spot fungus was long known as *Septoria ribis* Desm. The name of the perfect stage has recently been changed to *Mycosphaerella ribis* (Fckl.) Feltg.,[1] but for many years it had been referred to as *M. grossulariae*. It was found that this name was based on a quite different fungus from that causing the common leaf spot of *Ribes*.

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[1] Class Ascomycetes, Order Sphaeriales, Family Mycosphaerellaceae.

The *pycnidia* appear as small scattered brown to black dots, mainly on the undersurface of the leaf. The *conidia* are filiform, hyaline, two- to three-septate, 40 to 50 by 1.5 to 3 μ. The *perithecia* are produced on the fallen leaves during the fall and winter months. They are mainly hypophyllous, somewhat gregarious, spherical, 60 to 105 μ in diameter, with small papilliform ostioles. The *asci* are clavate to cylindrical, without paraphyses. *Ascospores* are slender, straight, one-septate, hyaline, 28 to 35 by 3 to 4 μ, the two cells about equal in size.

A closely related fungus, *M. aurea* (E. and E.) Stone, causes a similar leaf spot of flowering currants (Fig. 92). These spots are usually larger than those caused by *M. ribis,* and the conidia are longer. It has a restricted host range.

Hosts. *M. ribis* occurs on a number of species of *Ribes,* both cultivated and wild, but has not been reported as occurring on the ornamental species. The spot on these is caused by *m. aurea.*

Disease Cycle. The mature ascospores are discharged in the spring, usually in late May, and infect the green *leaves.* The leaf spots first appear 2 to 3 weeks later. The pycnidia appear in a week or so after the spots become plainly evident. The abundant conidia are forced out of the pycnidia during damp weather and renewed infection during the remainder of the summer. Fallen leaves furnish the inoculum for the spring infection. So far as known, the fungus is confined to the leaves.

Control. Application of fungicides for the control of anthracnose is usually sufficient for the control of leaf spot.

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Virus Diseases of Ribes

Although currants and gooseberries are not regarded as being subject to many virus diseases, several such have been described but are considered of minor importance.

Black Currant Reversion. The virus causing this disease has been called "currant reversion virus," "*Ribes* virus 1," and "*Acrogenus ribis* 4." The disease is usually referred to as black currant reversion disease.

History, Economic Importance, and Geographic Distribution. The disease was first noted in Holland and Germany about 1904 and in England about 1912. In England it soon became of sufficient importance to warrant quarantine measures, requiring inspection of bushes from which graft wood was to be obtained. It is the most important virus disease of black currants, a fruit crop extensively grown in Britain and the northern countries of Europe.

This virus disease occurs in all countries in Europe in which the black currant (*Ribes nigrum*) is grown commercially. It has not been reported from North America, probably because of the limited production of black currants in this country.

Symptoms. The most reliable early symptom of reversion is to be found in the peculiar venation of the leaves developed from the buds formed the previous year. The middle lobe of the leaf normally has five lateral veins extending out on each side from the midvein. In reversion leaves, there are three or four subveins in this lobe above the sinus. At a more advanced stage the leaves are seen to be reduced in size, the lobes are narrower and have a flatter base and a more coarsely rugose surface. The reverted leaves are a deeper green than the normal leaves during the growing season.

The *inflorescences* on the affected bushes tend to become longer, and the stalks of the individual flowers are also longer than normal. The sepals are more pointed, more highly colored, and hairy, while the petals are much narrower. While there is some setting of fruit, often the abnormal condition may lead to sterility.

Causal Agent. Grafting of diseased shoots on healthy bushes proved that it was caused by a virus which was not sap-transmissible. In 1927 it was found that the big bud mite, *Eriophyes ribis*, was an agent in the transmission of the virus in nature. Other vectors, especially aphids, have been suspected.

Hosts. Known to occur only on the European black currant (*R. nigrum.*).

Control. Roguing diseased plants before the crop is picked and use of sprays to control the big bud mite will prevent the spread in an established planting. New plantings should be from certified nursery stock.

Currant Mosaic. A virus disease of red currant (*R. rubrum*) was first reported by Hildebrand from the Hudson Valley in New York. Between 1935 and 1938 it had spread rather rapidly through this region and caused alarm among the growers.

The most striking *symptom* is the chlorotic patterns in the leaves. Irregular light green circular spots appear, mainly along the midrib and larger veins. Later, these spots enlarge and join, forming a clear, irregular band along the veins. It is unusual for the entire leaf to become chlorotic. The affected light-green areas gradually turn white. By July necrosis may set in, especially at the margin of the badly affected leaves.

Stunting of the canes occurs in advanced stages of the disease, and affected plants decline in vigor and fruitfulness and after several years may die.

Transmission of the virus has been obtained by inarching. Insect transmission is suspected, but has not been proved.

Other Virus Diseases. Posnetti, in 1952, reported on three virus diseases of *Ribes* which he regarded as new. He was able to transmit the virus to healthy plants in each case by bark grafting. The three were (1) *gooseberry vein banding* characterized by pale yellow, semitransparent banding of the primary and secondary veins. The virus was transmitted by inarch and saddle grafting and by aphids. (2) *Vein-pattern disease* of black currants, characterized by bright yellow bands, on primary and secondary veins. Transmission of virus was obtained by bark grafts. (3) *Black currant yellows*, characterized by young leaves with yellow flecking on fine veins followed by a yellowish-green mottle. By the end of June, segments of affected leaves are yellowish green. The yellow pattern is frequently accompanied by slight puckering of the interveinal areas. The virus was successfully transmitted by bark grafting.

The relationship of one of these diseases to the currant mosaic described by Hildebrand has not been determined, but the symptoms in some respects seem very similar.

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CHAPTER 12

DISEASES OF THE CRANBERRY

The Pilgrims when they landed in Massachusetts in 1620 found a low thornless bush with red berries growing over low swampy areas. Later they learned from the Indians that these berries were edible, and from that time to the present the "craneberry," as it was first called, became a prized American fruit. Until about 1816 cranberries were gathered from the wild bushes which grew abundantly in swampy areas where they were flooded in winter but drained during the summer months. The technique of cultivating cranberries was worked out about the middle of the past century. This consisted in selecting sites where the bushes could be flooded during the winter to protect them from extremely cold weather and to keep down insects.

The cranberry *Vaccinium macrocarpum* is native from Nova Scotia in Canada to North Carolina and westward to Wisconsin. The commercial crop is grown mainly in Massachusetts, Wisconsin, New Jersey, Washington, and Oregon. As many as 800,000 barrels are produced in an average year. The value of the crop in the United States is from 10 to 20 million dollars. The complicated technique required in growing cranberries has restricted the industry to a limited number of experienced growers and to areas where soil and water conditions are known to be suitable for successful production.

The only diseases of economic importance aside from false blossom are those affecting the fruit, and most of these are postharvest rots which appear in storage, but in some cases result from inoculation in the field.

In recent years the loss from fruit rots has been materially reduced, partly as a result of improved harvesting and packing methods, but mostly because of the fact that the bulk of the crop is now canned shortly after harvest before the rots become established.

Only those diseases causing appreciable loss in production or in storage will be treated.

References for all diseases are given on pages 478–479.

False Blossom of Cranberries

History and Geographic Distribution. The virus disease "false blossom" was first observed in Wisconsin and was so named because of the malformation of the floral organs. It was briefly described by Shear in 1911, but he stated at that time that it was known to Wisconsin growers long before that date. Since it was then known to occur only in Wisconsin and did not appear in other cranberry regions until a number of years later, it is reasonable to suppose that it originated in that state, probably on wild plants. In 1914 it was found in Massachusettes and later in New Jersey and the Pacific Coast region, but in all cases only on plants obtained from Wisconsin. By 1924 it was generally distributed over New Jersey and Massachusetts. It caused a very serious drop in production, and by 1928 false blossom threatened to wipe out the industry in Wisconsin, New Jersey, and Massachusetts.

Early attempts to find a causal organism were not successful, and it was concluded that some nutritional deficiencies were responsible, although there was evidence of the spread of the disease from plant to plant.

In 1927 Dobroscky proved that false blossom was caused by a virus transmitted by a leaf hopper *Euscelis striatulus* (*Ophiola striatula*). Starting about 1929 the U.S. Department of Agriculture began a study of the resistance of different varieties to false blossom. As a result of this study it was concluded that susceptibility or resistance to the disease was in fact conditioned by the relative preference of the vector for the different varieties as food plants. This work has been continued, and selections of vector-resistant varieties have been under test for a number of years.

As soon as it was determined that the leaf hopper was responsible for the spread of the disease, effective control measures were worked out so that the disease is now of much less importance than formerly.

Economic Importance. False blossom is considered the most important cranberry disease. No fruit is produced on infected plants, thus such plants are a total loss, since a plant once infected never entirely recovers. Bogs where the disease has become well established are rendered worthless. Recently losses have been greatly reduced by proper control methods.

Symptoms. Phyllody, or malformation of the *floral organs,* is the most conspicuous symptom of this disease. The flower *pedicels* become more or less erect instead of drooping, and the *calyx lobes* become enlarged, and take on a greenish leaflike appearance. The *petals* are short and broad with a reddish to greenish color. Diseased plants have a tendency to develop lateral branches from the latent axillary buds along the vine below the fruit buds. These laterals are slender and do not produce normal flowers. The whole effect of this growth is to give a witches'-broom appearance to the plant.

Causal Agent. The virus causing false blossom was named *Chlorogenus vaccinii* by Holmes. It is also known as *Vaccinium* virus 1 and Wisconsin false blossom virus. The virus has not been isolated, and little is known as to its physical and chemical properties. It is inactivated in the plant by heat at 42 to 43°C for 7 to 9 days, but many of the plants subjected to this temperature will be killed.

Hosts. The only natural host is the American cranberry (*Vaccinium macrocarpon* and the European species *V. oxycoccus*). Further details on host relations are given below.

Transmission. Transmission of the virus in nature is by the blunt-nosed leaf hopper, *Ophiola striatula* (*Euscelis striatulus*). This insect is very abundant in the cranberry bogs where the disease occurs and was suspected of being responsible for transmission before Dobroscky proved its role in the spread of the disease. She investigated a number of other leaf hoppers feeding on cranberry, but failed to get transmission. Artificial transmission by the use of juice from diseased plants was unsuccessful, but this was thought to be due to the difficulty of successful grafting. Kunkel was successful in transmitting the virus by the use of dodder. He first transmitted the virus to periwinkle and tomato from diseased cranberry plants and then from these hosts back to the cranberry. An interesting feature of these experiments was that the virus persisted in the dodder indefinitely without showing any symptoms. Moreover, it evidently multiplied in this host. By means of dodder transmission Kunkel was able to get infection on 28 species of plants in 10 different families. Phyllody was the most common symptom exhibited by these hosts. He concluded that the virus was not related to any other common virus, that it was probably not a mixture, and that possible "strains" existed. Kunkel, however, was of the opinion that the virus causing big bud on tomato was closely related to the false blossom virus since the symptoms on tomato when inoculated with the false blossom virus were similar to those described for big bud. No one to date has reported successful transmission of the big bud virus to cranberries.

Disease Cycle. The blunt-nosed leaf hopper, the only known vector, has only one brood a year. The eggs are laid in the bark in July and August and here remain over winter. They hatch in late May or early June. Most of the feeding, therefore, is during the months of June and July when infection probably occurs. The incubation period in the insect has not been accurately determined. The disease ordinarily spreads rather slowly from plant to plant. The period from inoculation to appearance of symptoms in the cranberry is remarkably long. In some cases plants infected one year show no evidence of the disease until the following year.

Plants once infected retain the virus indefinitely, although they are not killed. The cranberry sends out runners which root, and from these new shoots are sent up. Such shoots arising from runners of diseased plants always show the typical virus symptoms. Thus a large area in a bed may show diseased plants arising from a single infected mother plant.

Varietal Relations. No variety is known to be immune to false blossom. Of the varieties constituting the largest acreage, Howes is very susceptible, while Early Black and McFarlin are somewhat resistant. Shaw's Success is regarded as the most resistant, but this variety is not commonly grown. The breeding work done by the U.S. Department of Agriculture has resulted in the release of three named varieties, Beckwith, Stevens, and Wilcox, which are considered resistant because they are unpalatable to the leaf hopper and consequently escape infection.

Control. The control of false blossom is based on the control of the vector. Two methods of control have been advocated: (1) reflooding of the bogs shortly after the leaf hoppers have hatched and (2) spraying or dusting with effective insecticides. The timing of these operations and the choice of methods depend on the local situation. All diseased plants should be removed in so far as possible, but the intertwining of the plants in a bog often renders complete roguing difficult.

If new plantings are to be established, the selection of some of the new varieties resistant to leaf hopper feeding should be considered. (For references see page 478.)

Fruit Rots

The symptoms and effects on the fruit produced by the various species of fungi are so similar that they may be treated as a group. The severity of these diseases varies greatly in the different cranberry-growing regions and from season to season.

History. Attention was first called to the nature of cranberry diseases by Halsted, who published in 1889 on this subject. The most extensive investigation of these diseases was initiated in 1901 when Shear was requested by the American Cranberry Growers' Association to investigate the cause of fruit rotting in the eastern bogs. He, together with Stevens, continued these studies over the next thirty years. They identified the fungi concerned and made extensive studies on the conditions under which these diseases were likely to be of importance, both in the field and in storage. They contributed also to the nonparasitic conditions, such as sterile breakdown of the berries in storage, and to certain vine diseases which caused loss of plants in the field. Since cranberry growing is limited to a few concentrated areas, relatively few additional contributions have been made to the diseases of this crop. Below is presented a brief account of the principal fruit rots.

End Rot

Taking the country as a whole, end rot is the most important of the fungus fruit rots. The rot usually starts from either the blossom or the stem end, hence the common name. Aside from this it has no characteristics to distinguish it readily from the other rots. The fruit when attacked becomes softened and finally wrinkled. It usually appears in storage or late in the season, although the fungus is known to be present on the fruit in the field as early as the middle of July.

Causal Organism. The fungus causing end rot is an Ascomycete, *Godronia cassandrae* Pk.[1] The *apothecia* are produced on diseased twigs and fruit of the cranberry as well as several other species of *Vaccinium* and *Cassandra calyculata.* The *asci* borne in the small brown apothecia are cylindrical in form, 60 to 100 by 5 to 10 μ. The *ascospores* are hyaline, filiform, with six to seven septa, and measure 50 to 70 μ in length. The pycnidial stage is a *Fusicoccum* and is on the twigs or berries. The *pycnidium* is usually chambered but may be simple. The *spores* are elliptic to fusiform, hyaline, continuous, 10 to 12 by 2 to 3 μ.

Disease Cycle. The ascospores are discharged in the spring months, and spores from the pycnidia on the twigs also ooze at this time. A limited amount of infection in the field occurs during May and June, but the fungus is quiescent during the remainder of the summer, and little fruit rotting is observed until after harvest. It is typically a late-storage rot. This rot is prevalent in the colder sections (Wisconsin, Pacific northwest) and is of less relative importance in New Jersey, where the temperature is higher.

Early Rot

While primarily a *fruit* rot, this disease attacks all the aerial parts of the plant. It has also been called "blast," since under favorable conditions it may attack the *flowers* and *young berries*, causing them to shrivel and blacken. However, blast or blight may result from other causes, such as imperfect fertilization or from unfavorable weather conditions.

Causal Organism. The fungus causing this disease is *Guignardia vaccinii* Shear.[2] In many respects this fungus is similar to that causing black rot of grapes. The *pycnidia* appear as black dots on the infected area. The *spores* are hyaline, obovoid, 10 to 13 by 5 to 6 μ. A gelatinous, apical appendage similar to that of the apple blotch spores is sometimes present. The *perithecia* resemble the pycnidia in size and form. The *ascospores* are hyaline at first but appear yellowish or brown when old. They are short-elliptical and measure 13 to 16 by 6 to 7 μ.

Symptoms. On the half-grown fruit the lesions appear at first as minute light-colored spots. As the rot spot enlarges, concentric dark-colored rings may appear, but when the rot spreads rapidly, little coloration is evident. Berries attacked before they are three-fourths grown frequently shrivel and become covered with the black fruiting bodies of the fungus. In other cases, especially in the colder northern region, the fungus does not produce the rot until after harvest, although it may be present on the surface of the berries. Sometimes the fungus attacks the leaves, in which case it produces irregular reddish-brown spots with scattered spore-bearing pustules. If the whole leaf is invaded defoliation results.

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[1] Class Ascomycetes, Order Helotiales, Family Ceniangiaceae.

[2] Class Ascomycetes, Order Sphaeriales, Family Mycosphaerellaceae.

Early rot unlike end rot is much more prevalent in the southern cranberry-growing areas.

Bitter Rot

Glomerella cingulata (Stan.) Spaul. and V. Shrenk var. *Vaccinii* Shear[1] causes a field and storage rot of some importance, especially as an early rot on fruit from Massachusetts and New Jersey. The disease is very similar to bitter rot of apples. The morphology of the fungus and the disease cycle are much the same as of apple bitter rot. The symptoms of this rot are so similar to the other fruit rots of cranberry that unless the fruiting bodies of the fungus are present it may be mistaken for any one of the others. It is also erratic in its appearance from season to season. Hot periods during July and August with frequent rains are conducive to its development.

Rather indistinct brown spots appear on the *fruit*, usually during the time when the berries are nearing maturity or after harvest. Unless very moist conditions prevail, the fungus spreads throughout the tissues without the appearance of the fruiting bodies. When these are present they erupt, producing a mass of pink spores in acervuli. A rather soft rot results after the tissues are invaded. The perfect stage of the fungus has not been found in nature, but develops in culture.

The mode of overwintering is not known except that the fungus mycelium in the mummied berries remains alive for a long period. The fungus has been found fruiting on leaves, and since cranberries are evergreen plants, the spores from these leaf lesions may bring about infection during the season on new leaves and later on the fruit.

For further details of the morphology of the fungus see under Bitter Rot of apple.

Blotch Rot

In New Jersey one of the most common fruit rots is caused by *Acanthorynchus vaccinii* Shear.[2] This fungus has been reported from other eastern states but is of lesser importance in the colder northern bogs.

The rot cannot be distinguished from some of the other cranberry rots so far as symptoms are concerned. A small light-colored lesion first appears on the *fruit*, and sometimes concentric light and dark rings may appear, but this is not a constant character. Dark-colored blotches sometimes appear on the skin of the fruit, hence the common name. The entire fruit is invaded and finally destroyed.

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[1] Class Ascomycetes, Order Sphaeriales, Family Gnomoniaceae.

[2] Class Ascomycetes, Order Sphaeriales, Family Fimetariaceae.

The fungus causing the disease has no known imperfect stage but produces perithecia on the *fruit* and *leaves*. The globose to pyriform, subepidermal *perithecia* have a beak with black spines. The *asci* are clavate, 130 to 180 by 30 to 48 μ. *Ascospores* are brown at maturity, oblong, elliptical, densely granular, 27 to 36 by 12 to 20 μ.

The ascospores are discharged in the spring and, when they alight on the leaf, germinate to produce a lobed appressorium. From this an infection peg arises which penetrates the leaf, or a short germ tube grows out and enters the leaf through a stomate. It is assumed that a like method of infection occurs on the fruit, although this has not been accurately determined. (For references see page 478.)

Other Vine Diseases of the Cranberry

Most of the rot-producing fungi are present in the bogs on twigs when these start to develop in the spring, but for the most part are either weakly parasitic or entirely saprophytic in this stage. A few well-defined diseases are present at times on the leaves and shoots, causing various malformations on these organs. They are very sporadic in their appearance, but at times may cause severe damage to the plants.

Red Gall. This disease caused by *Synchytrium vaccinii* Thomas[1] is easily recognized. The disease appears just before the blossoms open. On the *buds*, *flowers*, and *young leaves* are numerous small red globular galls about ⅛ in. in diameter. These may appear on the young shoots also but are more scattered and not so conspicuous. The diseased shoots produce no fruit.

The fungus causing this gall disease belongs to a low order of the Phycomycetes. It produces a *resting spore*, or *sporangium*, located in the gall tissues. The content of this organ gives rise to a mass of *swarm spores* which are liberated by the rupture of the sporangial wall. The fungus does not produce a true mycelium. The motile spores are usually liberated in the spring, swim about in the water for a short time, and finally come to rest on the plant parts where they form a penetrating germ tube, thus gaining entrance to the host cells.

The formation of a new gall is brought about by the swelling of the infected cells and increased production of host cells surrounding these. The endospores remain in the galls throughout the season in a resting condition.

This fungus occurs on a number of hosts belonging to the heath family (Ericaceae).

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[1] Class Phycomycetes, Order Chytridiales, Family Synchytriaceae.

Red gall has been reported mainly from the eastern United States. It is erratic in its appearance and is of no great economic importance.

Rose bloom caused by *Exobasidium oxycocci* Rost[1] attracts attention because of the striking appearance of the rose-colored *leaves* produced as a result of the infection. The disease makes its appearance soon after the water has been drained from the bogs in the spring. The buds in the axils of the leaves are attacked by the fungus. Abnormal lateral shoots are produced bearing rose-colored leaves. These enlarged leaves crowded together on the shoot give a flowerlike appearance, thus giving rise to the common name of the disease. It has also been called "false blossom," but this term is used in connection with an entirely different disease.

Rose bloom is reported from Washington and Oregon as well as from the eastern bogs.

The fungus causing this disease belongs to the Basidiomycetes, but produces no distinct fruiting body. The *basidiospores* are borne on a loose mycelial mat on the surface of the diseased parts. The disease is of minor importance, although in some years it may cause considerable crop reduction.

Red leaf spot is caused by a fungus, *Exobasidium vaccinii* (Fckl.) Wor.,[1] very similar in many respects to the one causing rose bloom. It is confined mostly to the *leaf* where it produces more or less circular bright red spots on the upper surface of the leaf. On the lower surface the spots are covered with a powdery bloom which results from the production of the numerous basidiospores on the superficial hymenium.

The fruit occasionally is attacked, resulting in elevated, bright red spots on the green berries. The fungus also may grow down the leaf petiole and along the tender developing runners, where it causes swelling but not death of the *shoot*. In the Northwest this hypertrophied region often is invaded by another fungus which gives it a black appearance. This same fungus also follows the red spot on the leaves, causing shriveling of the leaves and defoliation.

Red spot is reported from all cranberry-growing areas in the United States but usually does not cause serious injury except under conditions of excessive moisture.

Black stem spot is caused by an Ascomycete, *Mycosphaerella nigromaculans* Shear. This disease has been reported from all areas where cranberries are grown. As noted above, it is often associated with red spot, especially in Washington.

The fungus gains entrance through the leaves, grows down the petioles, and forms elongated black spots which may completely girdle the stem. Defoliation and death of the stem follow the girdling.

The fungus produces fruiting bodies on the dead stems in the fall.

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[1] Class Basidiomycetes, Order Exobasidiales, Family Exobasidiaceae.

These develop into perithecia in the early spring, and the ascospores are discharged during rainy periods to bring about renewed infection which may continue throughout the growing season.

The imperfect stage of this fungus is a *Ramularia* similar to the imperfect stage of the strawberry leaf spot (*M. fragariae*).

The ellipsoid hyaline ascospores are almost equally two-celled, constricted at the septum, 23 to 30 by 3 to 4.5 μ. The upper cell is somewhat broader than the lower.

Sclerotinia tip blight caused by *Sclerotinia oxycocci* Wor. also causes a fruit rot called "hard rot," or "cotton ball." It occurs in the Pacific northwest and in Wisconsin. The fungus overwinters in the mummied berries which have fallen to the ground. From these sclerotia, small slender-stalked apothecia are produced in April or early May. These are produced beneath the bushes, and the spores are discharged during the time the new growth is developing. The tender shoots are infected, later turn brown and wither. On these dead stems the conidial stage of the fungus appears as a grayish powder. These conidia are produced during the blooming period of the plant and apparently infect the developing fruit at this time. The infected berries continue their normal increase in size all summer without showing any external evidence of infection, but when such berries are cut open they show a white cottony mass of mycelium in the seeds. The berries continue normal growth until shortly before ripening, when the fungus invades the entire berry, which then becomes filled with the cottony mass. This later forms a hard, compact black sclerotium from which the apothecia develop in the spring.

The disease is most abundant in seasons when prolonged wet periods prevail during the time when the apothecia mature and when frequent rains or fogs occur during the blossoming time. The presence of abundant mummied berries on the debris beneath the vines is also necessary, since it is only through these that the shoot-tip infection can occur. This explains why the disease is more severe in old than in new bogs.

Some *control* has been secured by sprays applied during the blooming period, but the erratic appearance of the disease rarely warrants the cost of control by this method. Clean harvesting of the fruit will reduce the chances of overwintering of the fungus. [H. F. Bain, Cranberry disease investigations on the Pacific Coast. *U.S. Dept. Agr. Bull.*, **1434** (1926).]

Physiological Diseases of Cranberry

Winter Flooding Injury. Cranberry bogs are flooded in winter to protect them from freezing. When the water is withdrawn in the spring and growth starts, it is often noticed that injury has occurred. In extreme cases death of some of the stems, leaves, and buds results. Other forms of injury are leaf drop, death of terminals, retardation of new growth from the terminals, death of some of the flower buds, and failure of the flowers to set fruit or stunting of fruit. Reduced oxygen supply in the flood water, especially after the bogs are frozen over, is regarded as the cause of this injury. The amount of dissolved oxygen depends partly on the depth of the flood water and partly on the photosynthetic activity of plant life in the bog water. Lack of oxygen "smothers" the plant by reducing the normal respiration which occurs in all functioning cells, and the most active tissues may thus be killed. Two methods of control have been advocated: (1) the flood water may be drained off after 8 to 10 in. of ice has formed, and the ice allowed to sink into the vines; (2) the vines may be frozen into the ice by adding water gradually until the ice is thick enough to protect without damage. This ice contains sufficient oxygen to permit respiration of the plants. Shallow flooding is also advocated, so that while the lower parts of the plants are in the water, the tops which are most active are in the ice.

Sterile Breakdown of Berries. The greatest loss in storage results from a type of rot similar in many respects to that caused by the rot resulting from fungus infections, but no causative organisms are present. The condition is similar to that occurring in many fruits when held too long in storage. The flesh of the berries softens, and the fruit shrivels. This type of rot is prevented in the early stage of storage by maintaining proper temperatures, but increases rapidly in spite of refrigeration after prolonged storage. Recent increase in processing the berries shortly after harvest has greatly reduced the loss from breakdown.

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CHAPTER 13

DISEASES OF THE BLUEBERRY

THE BLUEBERRY

The blueberry has a world-wide distribution, but until comparatively recently, most of this delicious fruit was obtained from the wild plants which grow in great abundance in abandoned fields or in open wooded areas where the tree growth is not too dense. They thrive on rocky hillsides, in swampy areas, or in open spaces in the dense forest. Within recent years superior, large-fruited varieties have been developed by selection and breeding, and the cultivation of blueberries on a large scale has become an established industry in the United States and Canada.

The blueberry belongs in the genus *Vaccinium* of the heath family, as does the cranberry. The "huckleberry," often confused with the blueberry, is closely related, but differs from the blueberry in that it has 10 rather large seeds while the numerous small seeds of blueberry species are hardly noticeable when the fruit is eaten. The separation of blueberries into definite species has been difficult on account of the extensive hybridization which has taken place in the wild. This can be understood by anyone familiar with the great variation one finds in picking the fruit. The two types generally recognized are the low and the high bush. The dominant low-bush blueberry is *Vaccinium angustifolium* generally encountered in New England and westward to Minnesota, while the high bush, *V. corymbosum* (*V. australe* Small), is found throughout the Atlantic Coastal plain from New England to Georgia and westward to Lake Michigan. The cultivated varieties of blueberry were derived mainly from the high-bush species. In the southern United States, the rabbit-eye blueberry, *V. asheri*, is common, and selections and breeding from this species have given rise to a number of cultivated varieties for the South.

Blueberries thrive only on acid soils, and heavy mulching with acid-forming materials such as leaves and sawdust is practiced where heavy acid humus is not naturally present. The acid-soil requirement is the main limiting factor in the geographical distribution of the blueberry. As is the case with the cranberry, propagation and spread of the blueberry are by an extensive growth of underground runners which grow through the humus close to the surface.

Most of the information on blueberry diseases recorded in the following pages was obtained from the excellent summary by Demaree and Wilcox published in 1947 [*Phytopathology*, **37**:487–506 (1947)]. In addition, M. T. Hilborn kindly furnished up-to-date information concerning the blueberry disease situation in the New England states during recent years.

References to all blueberry diseases are listed at the end of this chapter, rather than following each disease.

Leaf Rust of Blueberry

Two heteroecious rusts occur on blueberries. One known as "leaf rust" (*Pucciniastrum myrtilli*) has its alternate stage on hemlock (*Tsuga*), while the second, causing "witches'-broom," has species of *Abies* (fir) as its alternate host.

Geographic Distribution. *Leaf rust* is about as widespread as powdery mildew and occurs on most species of *Vaccinium* as well as other genera of the Ericaceae. It ranges from Nova Scotia to Wisconsin, southward to Florida and Arkansas, and in western North America from Alaska to New Mexico. It has been reported also from South America, Europe, Asia, and Japan.

Economic Importance. Where blueberries are grown near the hemlock in the New England states, serious defoliation occurs in some years. This is true also in some of the southern Atlantic states where it is not dependent on its alternate host, but may survive mild winters on the blueberry and thus start an early infection which may assume epiphytotic proportions by June. When defoliation occurs early in the season, the berries may shrivel and drop off. Markin reports that in 1931, where leaf rust and mildew were controlled by spraying the increased yield over the untreated plots was from 200 to over 320 per cent, but these experiments did not indicate the amount of damage which could be ascribed to the rust alone. Because of its sporadic appearance and the fact that often defoliation does not occur until late in the season, this rust usually is of no great economic importance.

Symptoms. Leaf rust is confined to the *leaf* of the blueberry. Discolored spots appear on the lower surface of the leaf. On such spots the yellow pustules of the uredial stage of the fungus appear during the summer months. Defoliation usually occurs rather late in the season but may start as early as July.

Causal Organism. The rust fungus causing this leaf disease is *Pucciniastrum myrtilli* (Schum.) Arth.[1] Pycnia and aecia are produced on the underside of the *leaves* (*needles*) of the hemlock (*Tsuga*). The cylindrical *aecia* appear in rows on each side of the midrib of the leaf. *Aeciospores* are globoid, 15 to 21 by 18 to 27 μ, with finely verrucose walls. The yellowish *uredia* appear on the lower surface of the *blueberry leaves.* They may be scattered or gregarious on the discolored spots. The *urediospores* are ovate or ellipsoid, 12 to 19 by 16 to 24 μ, with minutely echinulate walls. The one-celled *teliospores* are produced *within* the epidermal cells on the lower leaf surface, forming a platelike layer. They are oblong, 7 to 10 by 14 to 17 μ and nearly colorless.

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[1] Class Basidiomycetes, Order Uredinales, Family Melampsoraceae.

Hosts. As stated above, this rust attacks practically all species of *Vaccinium* as well as other members of the heath family. The hemlock (*Tsuga canadensis*) is the only alternate host so far as known.

Disease Cycle. In northern regions the fungus passes the winter in the telial stage in the leaves of the blueberry. These germinate in the spring, and the basidiospores are carried to the hemlock where the aecia are produced in the late spring months. The aeciospores sift from the aecial cups and are carried by air currents to neighboring blueberry bushes. The mode of infection has not been determined, but it seems to take place on the lower surface of the leaf. It has been proved by inoculation studies that urediospores may appear on the leaves as early as 9 days after the sowing of the aeciospores. According to Markin the rust often does not appear on the blueberry until fairly late in the summer, thus reducing the damage to the bushes.

In the South the disease cycle is different, for here the alternate host is rarely in close proximity to the blueberries. Demaree and Wilcox state that in Georgia in 1945 the rust was so severe on *Vaccinium ashei* by mid-June that the early-formed leaves were falling and new leaves were developing. They account for the sporadic outbreaks by assuming that there is regular overwinter survival of uredia on wild species of *Vaccinium.* Thus the urediospores would be produced as soon as growth started in the spring and would soon spread the disease to neighboring plantations.

Wherever the disease occurs, the urediospores are responsible for the spread of the rust throughout the area. Later in the season the teliospores are formed in the blueberry leaves, thus completing the life cycle. (For references, see page 490.)

Witches'-broom of Blueberry

The second rust disease of blueberry is called "witches'-broom" since the infection in the stems results in clusters of slender shoots arising from the enlargements caused by the invasion of the fungus.

Symptoms. The disease appears on the *shoots* of the blueberry and not on the leaves. Under natural conditions the swelling on the stem and the characteristic witches'-broom effect appear in the spring following infection during the spring months of the previous year. Since this rust has no uredial stage and the teliospores are borne in the epidermal cells, there is little external evidence of the fungus. The disease is found only in close proximity to woodlands where the balsam fir is present.

On the fir the *aecia* are found in the spring mostly on the lower surface of the *leaves* (*needles*). These when mature extend well above the leaf surface as cylindrical tubes. Pycnia appear on the upper surface before the appearance of the aecia.

Economic Importance. No great loss is experienced from this disease since the fungus does not spread from plant to plant of blueberry, lacking as it does a uredial stage. In the presence of infected firs the local loss may be extensive if seasonal conditions are favorable. An infected bush becomes entirely useless since the fungus is perennial in the blueberry stem. On the whole this disease is regarded of minor importance.

Cause and Disease Cycle. The heteroecious rust *Pucciniastrum goeppertianum* (Kühn) Kleb.[1] causes witches'-broom. The pycnia and aecia are found on species of *Abies*. The *pycnia* occur on flecked spots on the upper surface of the leaf. The cup-shaped to cylindrical *aecia* are mostly hypophyllous on yellow spots. *Aeciospores* are broadly ellipsoid, 10 to 18 by 18 to 30 μ.

On the blueberry the fungus is systemic, forming a witches'-broom. The uredial stage is wanting. *Telia* are caulicolous, forming a continuous layer around the stem with a polished reddish-brown surface. The *teliospores* are formed in the epidermal cells in a closely pressed layer. They are prismatic, four-celled by vertical septa. They measure 10 to 14 by 18 to 30 μ.

On species of *Vaccinium* the disease has been reported from Newfoundland south to Pennsylvania in eastern North America and from Alaska south to Colorado and northern California. It is known to occur also in Mexico, Europe, and Japan.

According to Wilcox the teliospores from blueberries when suspended above the fir tree branches under moist conditions germinated and produced *basidiospores* in 2 days, and aecial pustules appeared 12 days later. The maximum spore production was reached in 2 to 3 weeks after the appearance of pustules on the fir needles. She found that aeciospores from the fir when sprayed on young blueberry plants would produce witches'-broom in 3 months, but that growth was retarded until cool weather in the fall. Under natural conditions witches'-broom generally appears in the spring, as a result of infection the previous year, thus giving an incubation period of approximately one year. The lack of the uredial stage in this rust restricts a wide spread of the disease on the blueberry, but the fact that it is perennial in this host increases the chance of infection of neighboring fir trees.

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[1] Class Basidiomycetes, Order Uredinales, Family Melampsoraceae.

Infection on the fir results from basidiospore inoculation. This occurs when the diseased blueberry bushes are in close proximity to the fir, as would occur when wild infected plants were growing in wooded areas.

Control. Elimination of diseased blueberry plants is rendered easy by the conspicuous witches'-broom, thus preventing reinfection of neighboring fir trees. It is obvious that where the disease is commonly found on fir trees, arising from infected wild blueberry plants, the grower should establish his plantation at some distance from the firs. (For references and for general control see page 490.)

Blueberry Blossom and Twig Blight

History and Geographic Distribution. The fungus causing this disease, *Botrytis cinerea*, has long been known as a blossom and twig blight organism on many other hosts. Rotting of a number of fruits is brought about by this mold (see Gray Mold Rot of strawberries). The first account of the disease of blueberries was by Markin, who in 1931 called attention to an outbreak of twig and blossom blight in Maine in 1930. In 1954 Pelletier and Hilborn presented a comprehensive study of this disease in which they called attention to serious losses in Maine in the 1951 season. It was also reported as the most serious disease of blueberries in Washington.

The disease as a twig and blossom blight has been reported from the New England states and the maritime provinces of Canada in eastern North America and from Washington, Oregon, and California in the northwestern United States. It is sporadic in its appearance, usually being of importance only in seasons of rainy or foggy weather during the prebloom and blooming period. It is not common in regions not subject to these conditions.

Causal Organism. The disease is caused by the common gray mold fungus, *Botrytis cinerea* Deb.[1] No cross inoculations have been reported indicating whether or not the blueberry fungus is a special strain of this species. Whetzel was of the opinion that the perfect stage of this fungus (*B. cinerea*) belonged in the genus *Botryotinia* and named it *B. fuckeliana*, but only on the evidence of the development of the perfect stage in culture. A morphological description of this fungus is given under the discussion of the Gray Mold of strawberries (page 427).

Hosts. *B. cinerea* as a fruit rot and blossom and twig blight has a very extensive host range. As a blight on blueberries it has been reported on most species of wild and cultivated high- and low-bush blueberries.

Disease Cycle. The fungus mycelium winters in the blighted *twigs*. When temperature and humidity conditions are favorable, the fungus will produce conidia on these blighted twigs in 24 to 48 hours. Thus there is abundant inoculum in the spring during the period when the flower clusters and tender shoots are most susceptible. These spores are dusty in character and are borne by air currents to the blossom cluster where they are able to bring about infection by direct penetration of the germ tube. They are capable of infecting if high humidity prevails for 4 or 5 days. They are also capable of infecting through wounds on the older twigs. From the infected blossoms the fungus grows down into the *shoots* to start twig blight. This may originate also from infected wounds. *Leaves* are infected when they come in contact with the blighted twigs or flower clusters. The twigs and blossoms soon become covered with the gray masses of conidia, and further infection may occur during subsequent rainy or foggy weather. It has been found that high humidity over a comparatively long period is necessary to bring about development of epidemics of the disease. Very little infection occurs late in the season, but the mycelium persists in the blighted twigs throughout the year.

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[1] Class Fungi Imperfecti, Order Monilales, Family Moniliaceae.

The optimum temperature for spore germination is between 18 and 29°C, with maximum mycelial growth in culture at 23°C (73°F). Both germination and mycelial growth are limited when temperatures approach 90°F. Pelletier and Hilborn demonstrated that a much greater infection occurred when a peat mulch is used over that of a sawdust mulch. They ascribed this to the water-holding capacity of the peat. Nitrogenous fertilizers applied in the early spring tended to increase infections more than those applied about the full-bloom period.

Control. Pelletier and Hilborn found that three applications of either ferbam (Fermate), Orthocide, or Phygon gave good control. Some of the other organic fungicides, either as dusts or liquid sprays, also gave satisfactory control. The applications were made at prebloom, full bloom, and after the fruit was set.

Cultural practices such as the use of sawdust rather than peat and avoiding early applications of fertilizers containing high nitrogen might aid in control.

Powdery Mildew on Blueberries

Powdery mildew is the commonest and most widespread disease of blueberries, occurring as it does from Maine to Florida and westward wherever blueberries are found. Usually it is not a destructive disease, although in seasons favorable for its development—hot and dry—it will cause extensive defoliation.

The fungus causing the disease is *Microsphaera alni* DC[1], one of the surface mildews which appears on a great variety of unrelated plants. Like the powdery mildews of grape, gooseberry, and apple, it belongs in the family, Erysiphaceae. The "oidium" (imperfect) stage forms chains of oval, hyaline *conidia* on separate *conidiophores*, thus giving the powdery appearance to the mildewed leaf. The small

[1] Class Ascomycetes, Order Perisphoriales, Family Erysiphaceae.

black *perithecia,* barely visible to the naked eye, may be seen scattered over the weft of mycelium later in the season. They are without ostioles (cleistocarpic), and the *appendages* are dichotomously branched and recurved at the tips. The *asci* number two to eight, and each ascus contains four to eight *ascospores.*

Injury to the diseased plants comes from the devitalizing effect of the fungus on the leaf, often resulting in defoliation by midseason. The fungus shows considerable variation in symptoms on different varieties. On some the fungus is confined to the upper surface, forming a compact layer or patches of mycelium. On others the hyphae are inconspicuous and confined to the undersurface of the leaf, with reddish areas where the fungus is attached.

Variation in susceptibility of cultivated varieties is marked. Bergman reported in 1939 that in Massachusetts, Pioneer, Cabot, and Wareham were most susceptible, Concord, Jersey, and Rubel were intermediate, while Stanley, Rancocas, Harding, and Katherine were highly resistant.

Control. While powdery mildew is common in many sections of the country, few experiments have been made on control. It is probable that in most seasons special control methods are not necessary. Judging from experience with powdery mildews on other hosts, it would appear that sulfur dusts would give control.

Blueberry Cane Canker

The cane canker disease is of considerable economic importance in the southeastern United States on cultivated blueberries. It is commonly present on the high-bush blueberry in North Carolina, and on rabbit blueberry in Alabama, Florida, and Mississippi.

Demaree and Wilcox state that the disease was first observed in North Carolina in 1938. Their study of the disease, reported in 1942, showed that it was rather widely distributed in the southeastern states and was caused by an Ascomycete which they named *Physalospora corticus.*

Symptoms. As the name indicates, this is primarily a disease of the *canes.* The first appearance of new infections is in the late summer or early fall, when reddish, conical swellings appear in the current-year shoots. Pycnidia of the fungus usually develop on the surface of these conical growths. In the second year on varieties such as Cabot and Pioneer the reddish color is lost and the lesions enlarge and become fissured. The surface takes on a rough blistered appearance and turns gray. The fungus may penetrate into the xylem, causing some discoloration but no necrosis of the wood. By the end of the second year the older portions of the canker are black, rough, and deeply fissured (Fig. 93). The final effect is the girdling of the canes and final dying. On some varieties the effect on the bark cortex is not so extreme, and the only evidence of the disease may be slightly swollen, uneven grayish areas with little bark cracking. The fruitfulness of the bushes is naturally impaired by this disease.

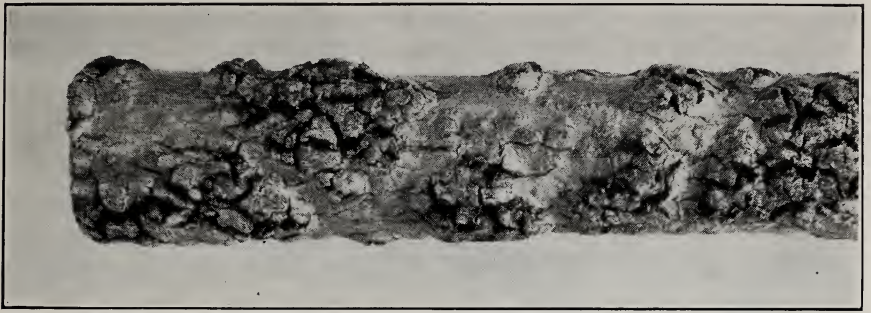

FIG. 93. Cane canker (*Physalospora corticis*) of blueberry. (*After Demaree and Wilcox.*)

Causal Organism. The fungus causing cane canker is *Physalospora corticus* Demaree and Wilcox.[1] The *pycnidia*, formed in the summer, are found in the margin of the cankers. They are black, conical to subglobose, measuring from 90 to 208 μ high and 60 to 165 μ wide. The *pycnospores* are fusiform, elliptical, nonseptate, hyaline, averaging 35 by 9 μ. Small rod-shaped spores have also been observed occurring in smaller pycnidia, but their relation to the disease is not known.

The *perithecia* are produced much more abundantly than pycnidia. They can be found in late summer in the recently invaded bark tissue. They are conical in shape, averaging 280 high by 200 μ wide with an ostiole forming a short beak. They resemble the pycnidia. The *asci* formed at the base of the perithecium push up through a paraphysate-like mass of anastomosing hyphae which was previously formed in the young perithecium. Usually only about 4 to 12 asci are formed in each perithecium. The *asci* are clavate with the wall thickened at the apex. The *ascospores* are hyaline, ellipsoid to fusoid, nonseptate, and average 30 by 12 μ.

Pathogenicity tests by Demaree and Wilcox proved that typical symptoms could be produced but that these did not appear until the year following the inoculation. Perithecia and pycnidia were formed on these inoculated plants, and the fungus was recovered from the invaded cortical tissue.

Disease Cycle. Only a limited amount of information is available as to how and when infection occurs in nature. The abundance of the perfect stage of the fungus on the cankered canes could well furnish inoculum for infection. From the location and distribution of the new infections, it is probable that the fungus gains entrance through the lenticels. An interesting but unexplained observation by Demaree and Wilcox was that cankers first appear on the side of the shoot directly exposed to the sun. After invasion the spread of the fungus is mainly through the cortex, which is not immediately killed.

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[1] Class Ascomycetes, Order Sphaeriales, Family Pleosporaceae.

Control. The disease is not easily controlled in old plantations in which it has become established. Selection of resistant varieties seems to be the best solution. Such very susceptible varieties as Cabot and Pioneer should be avoided. Disease-free plants should be selected in planting a new area, and these should not be planted near infected bushes in southern areas.

Minor Diseases of Blueberry

Leaf Spot Diseases of Blueberry. Various fungi have been described as causing leaf spots on species of *Vaccinium.* Demaree and Wilcox in 1947 recorded a serious leaf spot disease of cultivated blueberries in North Carolina and stated that it was not known to occur outside this state (Fig. 94). It appears to be confined to one species of the high-bush blueberry *Vaccinium anstrals.* The fungus was named *Dothichiza caroliniana* Demaree and Wilcox.

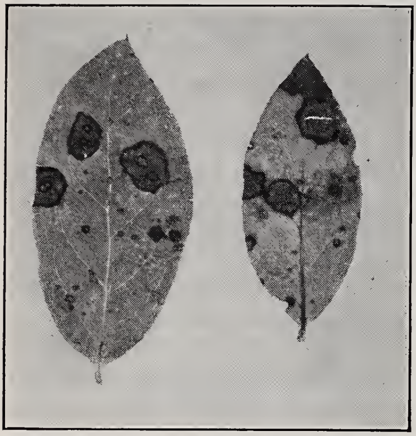

Fig. 94. *Dothichiza* leaf spot on blueberry. (*After Demaree and Wilcox.*)

Phyllostictina vaccinii Demaree and Wilcox causes small circular spots, grayish in the center and surrounded by a zone of brownish tissue. Usually only one pycnidium forms on a spot, thus resembling *P. solitaria* on apple leaves (see Apple Blotch). It resembles this fungus also in the production of pycnidia with pseudoparenchymatous tissue and the presence of gelatinous spore appendages.

Gloeocereospora inconspicua Demaree and Wilcox causes a leaf spot similar to that caused by *Dothichiza.* Defoliation caused by this fungus is sometimes serious, but on the whole it is of minor importance. It has been found mainly on high-bush blueberries in North Carolina and Maryland.

Mummy Berry Disease. A die back of shoots and hard rot of fruit results from the attack of *Sclerotinia vaccinii-corymbosi* Reade [*Monolinia vaccinii corymbosi* (*Reade*) Honey] if wet spring weather prevails. It is regarded as one of the most destructive diseases of the high-bush blueberry in years when present, but a number of years may pass without any evidence of diseased plants. This disease has been reported in blueberry plantings from Maine southward to Maryland. It was found on the rabbit-eye blueberry (*V. ashei*) in Mississippi and North Carolina, but appears to be rare in northern United States. The imperfect form of the fungus, a *Monilia*, blights the shoots and blossoms, often extending down the stem to the ground line. The fungus also attacks the fruit, which becomes dry and hard as the result of the formation of sclerotial masses. This characteristic is responsible for the common name "mummyberry." On the fruit, blossoms, and shoots, the gray *sporodochia* develop during damp weather. The monilioid *spores* are 19 to 29 by 14 to 26 μ.

The perfect stage of the fungus is a typical *Sclerotinia* apothecium which develops on the overwintering mummied fruit. As in brown rot of stone fruits, the ascospores are discharged in clouds in the spring and thus infect the developing shoots. The *Monilia* spores are carried by air currents and thus may spread the disease throughout a planting. There is no information available as to whether the initial infection may arise from conidia developing on overwintered diseased bushes.

Bacterial Canker of Blueberry. In 1953, Stace-Smith, Wooley, and Vaughan reported the occurrence of a new canker disease of blueberry (*Vaccinium australi*) in western Oregon which they claimed was caused by a species of *Pseudomonas.* This disease so far has not been reported from other blueberry-growing regions. The disease appears in January or early February as definite reddish-brown to black cankers which may extend from a few millimeters to the entire length of the stem of the canes produced the previous growing season. The buds in the cankered area are killed. Severe losses occur on susceptible varieties such as Jersey, Atlantic, Schammel, Coville, and Evelyn. The disease may be severe enough to kill young plants and destroy over one-half of the one-year-old stems of older plants. Vaughan and Boller reported that two applications of Bordeaux mixture (8-8-100) in October and November resulted in a substantial reduction in the number of diseased plants.

Control of Blueberry Diseases

Extensive experiments on blueberry disease control conducted by the Maine Agricultural Experiment Station workers have resulted in recommendations for the control of most of the serious diseases of this crop. They recommend:

1. Pruning blueberry plants by periodic burning. This does not control witches'-broom, but the latter may be partly controlled by destroying diseased plants with various weed killers.

2. Dusting with either 20-20-60 copper lime dust (calcium arsenate, monohydrated copper sulfate, and hydrated lime) or with 10 per cent ferbam dust during the first crop year. The first application should be made when most of the blossoms have dropped, with a second 10 to 14

days later. Where the leaf diseases are serious it may be desirable to make one application the second crop year, 10 to 14 days after blossom drop.

Virus Diseases of Blueberry

Blueberry Stunt. This virus disease is reported to occur in damaging amounts in New Jersey, North Carolina, and Michigan on high-bush blueberries. No variety is known to be immune, but Rancocas is highly resistant.

The *symptoms* consist of dwarfing of bushes, reduction in size of leaves, and yellowing of leaves during the summer, followed by a brilliant red during late summer and early fall. Berries on diseased bushes are small, with an unpleasant flavor. The disease most frequently appears on individual bushes scattered through a field and in adjacent bushes in a row.

The manner of natural spread is not known, but the virus can be transmitted to healthy plants by budding or grafting. Cuttings from diseased bushes show the stunt disease.

Basal shoots of affected bushes rarely reach a height of 3 ft, but usually do not exceed 1 ft.

The only method of control advocated is roguing, and in this process care should be taken to remove the entire bush so that no sprouting can occur.

Ring Spot of Blueberry. In 1954 Hutchinson and Varney described a virus disease occurring on the high-bush blueberry (*Vaccinium corymbosum*) in New Jersey, principally on the Cabot variety.

The symptoms are most conspicuous in the fall when the leaves of diseased plants show red rings or jagged oakleaf patterns characteristic of other ring spot virus diseases. Small red spots are evidence of the inception of the disease in the spring. Rings may also be seen on the stems throughout the year.

The virus was transmitted by whip grafting to 2-year-old virus-free Cabot plants, which showed distinct symptoms of the disease within 5 months.

The disease spreads rather rapidly in the field, but no vector has been discovered. The disease is of minor importance, since it is largely confined to the Cabot variety, which is no longer grown to any extent in the area where the disease occurs. Furthermore, it does not appear to cause serious damage even when present.

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